A longitudinal, controlled study of developmental enamel defects in preterm children

by

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Statement of Authenticity

The research work presented in this thesis is to the best of the candidate's knowledge and belief, original, except as acknowledged in the text. The material has not been submitted, either in whole or in part, for a degree at this or any other university.

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ABSTRACT

This controlled investigation is the first longitudinal study available on the developmental enamel defects and caries in the preterm children. In all, a total of 53 children participated, consisting of 26 very low birth weight (VLBW) preterm children (12 males, 14 females),and 27 full-term control children (13 males, 14 females) matched for age, sex, race and S.E.S. The preterm children recruited had a mean birthweight of 969±243g and a mean gestational age of 27±1.9 weeks whilst the control children had a mean birthweight of 3396±395g and a mean gestational age of 39±1.6 weeks. The mean age of all participating children at baseline examination (Exam I) was 29.8±4.5 months. These children were monitored for a mean period of 22.4±2.9 months (at 95% C.I.) and examined annually.

The results demonstrated higher prevalence of developmental enamel defects in the very low birthweight (VLBW) preterm children compared to full-term controls. The author found at Exam I, II and III prevalence rates of 88.0%, 94.7% and 96.0% in the preterm children, in contrast to 40.0%, 54.5% and 45.0% in the control children respectively (P-values<0.05). Not only were there more preterm children affected but the number of affected teeth in these children were markedly increased. Percentages of teeth affected at Exam I, II and III were 17.8%, 23.0% and 38.3% in the preterm children, in comparison to 3.4%, 5.5% and 5.0% in the control children respectively (P-values<0.00001).

The mean d.m.f.t. scores at Exam I, II and III were 0, 0.6±1.4 and 0.8±1.5 for the preterm children, in comparison to 0, 0.5±1.5 and

1.4±3.2 in the control children respectively (P-values>0.05). Judging caries prevalence using the d.m.f.t. scores may not be accurate because of the inflated d.m.f.t. scores for the control group as a consequence of 4 control children with high caries rate. Caries in the control group were found only in these 4 high caries risk children with the rest of the control group not affected by caries. It may be more pertinent to consider caries prevalence based on either the percentages of children with caries experience or the percentages of caries-free children. At Exam I, II and III the percentages of children with caries experience were 0%, 21% and 28% in the preterms, in comparison to 4%, 9% and 20% in the control children respectively (P-values>0.05). However, the differences between the numbers of preterm and control children experiencing caries did not reach statistical significance, as a result of the small numbers of children involved.

Analyses of caries risk factors investigated in this study found enamel defects to be the only underlying predisposing caries risk factor significantly associated with caries (P-value<0.00001). *Strep. mutans* score did suggest a possible association in the control children. Other caries risk factors considered did not reveal any association (P-values>0.05). These included brushing frequency, fluoride exposure, daily frequency of sugar intake and plaque score.

Of interest also was the fact that caries affected the preterm children of all social classes indiscriminately, rather than favouring lower social classes as in the full-term control group. This finding together with the highly significant (P-value<0.00001) association of enamel defects and caries in the preterm have important clinical implications. It would suggest that the underlying enamel defects present in the

preterm children obviate the effects of routine caries preventive measures including dietary and oral hygiene habits usually practised by the higher social classes. These measures would have been effective otherwise, in the normal situation where the children were unaffected by enamel defects. Preterm children with a high prevalence of enamel defects (resulting in an increased caries risk) would therefore require more comprehensive professional dental services.

Furthermore, this study noted a predilection for caries to occur in the posterior teeth of the preterm children. Dental caries was most frequently found on primary second molars (18% of maxillary and 10% of mandibular), followed by the first maxillary molars (4%) and with caries on the maxillary primary first molars, maxillary incisors and maxillary canines at the same frequency of 2%. The primary mandibular canines and incisors were not affected by caries.

Finally, caution is needed in interpreting all the above findings owing to the small number of children available for this longitudinal study. These findings await large scale studies for confirmation. Recommendations for future longitudinal studies include longer periods of follow-up, a larger number of children and shorter intervals of recall.

CHAPTER 1 INTRODUCTION AND LITERATURE REVIEW

INTRODUCTION

Advancement in medical science and biotechnology coupled with a higher standard of neonatal care have given many preterm infants the impetus to cope with life in a harsh environment outside the uteri, even as early as twenty-four (24) weeks after conception.

Improvements are observed in not only the survival rate but also the morbidity rate of these infants. With this come an ever growing number of preterm infants making up the paediatric population. Presently, preterm and low birthweight infants comprise approximately 6% of all live births (Yu, 1990). As these preterm infants make up a significant portion of the paediatric population, an increasing number of researchers and clinicians are devoting their resources to the field of prematurity. Also, preterm infants particularly the very low birthweight (VLBW, <1500 grammes) infants are considered to be at high risk for developmental problems. Consequently, much emphasis in the medical literature has been given to the development and needs of preterm infants.

In the dental literature however, although studies have reported on the higher prevalence of enamel defects in the preterm population, no research had been done looking at the long term sequelae and clinical implications of enamel defects suffered by the preterm children.

The existence of a world-class preterm care facility at the Mater Mothers' Hospital and the long term association of the Paediatric Dentistry Clinic of the University of Queensland with this facility have provided an excellent opportunity to follow-up dental research in the preterm children.

Hypotheses:

1. Preterm children suffer a higher prevalence of developmental enamel defects than full-term children.
2. These developmental enamel defects predispose preterm children to higher caries risk compared to full term control children.

Aims of The Study

The aims of this study are to:

(a) Document the occurrence of developmental enamel defects as well as other concurrent oral findings in a group of very low birthweight, (<1500g) prematurely-born children compared to a control group of matched healthy full-term children born at the same hospital at approximately the same period.

(b) Determine the importance of these enamel defects as a caries risk factor in preterm children.

A longitudinal study was performed for the following reasons:

(i) Dental caries takes time to develop, and it may be necessary to examine susceptible tooth surfaces over a period of time to determine if and when they become carious.

(ii) Once hypoplastic enamel becomes carious, it may be difficult to determine its original state, hence it is necessary to examine and document all original enamel states.

LITERATURE REVIEW

1.1 PRETERM INFANTS

1.1.1 Definition

Preterm or prematurely-born infants are those born before thirty-seven(37) completed weeks of gestation (WHO, 1977). This is in contrast to full-term infants who are born between thirty-seven(37) to forty-two(42) weeks gestation with birthweights averaging 3333 grammes(g).

1.1.2 Survival rates

In developed countries, preterm and low birthweight infants comprise about 6% of all live births but account for about 70% of neonatal deaths (Yu, 1990). The survival rates of preterm infants are very much dependent on gestational ages and birthweights. Birthweight of a preterm infant usually varies directly with gestational age. The higher the gestational age, the larger the birthweight. The percentage of preterm infants surviving range from a high of 98% with birthweights of 2000 - 2500g to a low of 26% for birthweights of 750g (Tudehope *et al*, 1983). More recently, the number of surviving neonates weighing between 700 - 799g has been reported to be at 56%, with even 10% of neonates weighing between 500 - 599g being able to survive (Yu, 1990). Furthermore, the number of survivors weighing below 1000g and not being crippled with major disabilities has risen to 88% (Yu, 1990).

1.1.3 Aetiology of preterm births

The aetiology of preterm birth is multifactorial. Nonetheless, in about

25% -50% of cases, the causes are unknown.

Possible causes and associations reported with prematurity include multiple pregnancies, alcohol consumption, smoking, low pre-pregnant weight, extremes of maternal age, history of infertility, previous termination of pregnancy, premature rupture of membranes, chorioamnionitis, abruptio placentae, pre-eclamptic toxaemia, threatened abortion and negative attitudes to pregnancy (Yu, 1989).

1.1.4 Medical complications in preterm births

The mortality and morbidity suffered by preterm infants are largely due to the underdevelopment of many of the organ systems at the time of preterm births. **Table 1.0** lists the medical complications encountered in preterm births. The complications are best looked at according to different organ systems.

1.1.4.1 Breathing system

(a) Perinatal asphyxia

Perinatal asphyxia occurs more frequently with preterm birth. Management would involve personnel expert in neonatal resuscitation to establish a clear airway (free from meconium, for instance) with adequate ventilation and satisfactory circulation.

(b) Hyaline membrane disease

The immaturity of the lungs and lack of surfactant frequently result in hyaline membrane disease (H.M.D.). Antenatal steroid therapy, avoidance of perinatal asphyxia, constant monitoring with appropriate oxygen and ventilatory therapy and lately where needed, surfactant replacement therapy are very effective in reducing the incidence and severity of respiratory failure and death from H.M.D.

TABLE 1.0* MEDICAL COMPLICATIONS OF PRETERM INFANTS

ORGAN SYSTEMS INVOLVED	PROBLEMS/COMPLICATIONS
Respiration	Perinatal asphyxia, hyaline membrane disease, recurrent apnoea, pneumothorax, pneumonia, bronchopulmonary dysplasia.
Immunity	Extreme susceptibility to infections
Metabolism	Hypothermia, hypoglycaemia, osteopenia, rickets, hyperbilirubinaemia
Blood	Anaemia, intracranial haemorrhage
Heart	Patent ductus arteriosus
Kidney	Fluid and sodium imbalance
Gut	Feeding difficulty, necrotising enterocolitis
Psychosocial concerns	Preterm stress

*Modified from Seow (1992)

Oxygen therapy which is often used to reduce the effects of H.M.D. in preterm infants is not without risk. Hyperoxia leads to retinolental fibroplasia which in turn causes blindness. On the other hand, hypoxaemia may result in brain damage or even death.

(c) Bronchopulmonary dysplasia

Preterm infants with severe respiratory disorder (e.g. H.M.D.) treated with prolonged endotracheal intubation, mechanical ventilation and a high inspired oxygen concentration may develop bronchopulmonary dysplasia (B.P.D.). B.P.D. is a condition with chronic respiratory distress and associated cystic changes of the chest and increased risk of superimposed infection. Nonetheless, the long term outlook is usually good (Yu, 1990).

(d) Recurrent apnoea is usually the consequence of inadequate development of the neurological control of breathing and the respiratory apparatus. However, other causes such as infection, periventricular haemorrhage or metabolic disorders may also cause recurrent apnoea. In affected infants, continuous electronic monitoring of the heart and breathing rate are required.

1.1.4.2 Immunity

These neonates are very prone to infections due to their under-developed humoral and cellular immune system, and the lack of maternally transferred immunoglobulin (Yu, 1990). Clinical features of infections are non-specific and include lethargy, diarrhoea, apnoea, jaundice, pallor, unstable temperature and refusal of feed. If an infection is suspected, cultures from blood, urine, nose, throat, umbilicus or even cerebrospinal fluid may be required. Effective infection prevention includes

scrupulous attention to hand washing for persons involved in handling infants.

1.1.4.3 Metabolism

(a) Thermal instability:

In preterm infants, excessive heat loss and diminished heat production lead to inadequate thermal regulation. Cold injury or stress increases mortality rate and reduces growth rate. These infants thus need to be placed in a thermoneutral environment.

(b) Disorders of calcium metabolism:

Deficiencies of calcium and phosphorus stores aggravated by poor oral supply and absorption are important in the aetiology of osteopenia and rickets in the preterm infants. Oral calcium and phosphorus supplementation are required.

(c) Hypoglycaemia:

Inadequate glycogen stores increase the tendency for hypoglycaemia in preterm infants. Symptoms which may or may not be present include lethargy, jitteriness, apnoea and convulsions. Routine monitoring of blood glucose following delivery at 4 to 6 hourly intervals are necessary. Hypoglycaemia is treated with glucose infusions until normal levels are maintained.

(d) Hyperbilirubinaemia:

A preterm infant should also be reviewed daily for clinical signs of jaundice and if this is more than a mild jaundice, a blood serum estimation is needed. Serum bilirubin need not be high in preterm infants for bilirubin encephalopathy

(kernicterus) to set in. Management of jaundice generally involves phototherapy (at bilirubin level of 150 μ mol/l) and exchange transfusion (at level above 200 - 300 μ mol/l) (Yu, 1990).

1.1.4.4 Blood

(a) Anaemia commonly develops due to circulating immature red blood cells and may require repeated small quantities of blood transfusion. Anaemia may also be caused iatrogenically by repeated blood sampling. Iron or sometimes folate supplements are introduced starting at 6 to 8 weeks of age to combat late anaemia with rapid post-natal growth.

(b) Hypoprothrombinaemia, liver dysfunction or infection contribute towards haemorrhagic diseases including intracranial periventricular haemorrhage to which preterm infants are more prone.

1.1.4.5 Heart

Ductus arteriosus remains patent in preterm infants and usually closes spontaneously only as full term approaches. Pharmacological closure with indomethacin therapy or surgical ligation may be indicated (Kliegman, 1990) if the severity of the patent ductus is sufficient to result in congestive cardiac failure, respiratory distress or ventilator dependence.

1.1.4.6 Kidney

Fluid and sodium imbalance in addition to the inability to conserve bicarbonate are problems associated with immature kidneys.

1.1.4.7 Gut System

(a) Milk feeding is extremely difficult due to poor suckling, delayed gastric emptying, regurgitation of feeds and poor intestinal transit.

(b) Necrotising enterocolitis is a common serious complication requiring gastric aspiration, intravenous fluids, antibiotics and less frequently, bowel resection.

1.1.4.8 Psychosocial concerns.

(a) Detachment from parent early in life following a preterm birth and the "unfavourable" nursery care environment compared to intra-uterine life all add towards increasing psychological stress in the preterm infant. Early parent-infant interaction and participation of parents in care-taking activities may reduce stress and enable the development of a normal bond between parents and infants. Psychosocial needs of parents and infants should be recognised and effectively managed to include appropriate parental counselling.

(b) Preterm infants are considered at particular risk for later child abuse and neglect (Yu, 1990) for a variety of psychosocial reasons related directly or indirectly to their premature births.

1.2 Enamel defects in preterm children

1.2.1 Introduction

Developmental enamel defects can result from various disturbances during amelogenesis (McDonald and Avery 1983). Enamel defects may manifest clinically as decreased enamel thickness called enamel hypoplasia (arising from deficiencies in enamel matrix formation) or as abnormalities of enamel translucency known as enamel opacities (arising from interference in calcification and maturation of the enamel) (Seow 1986).

Studies have shown enamel formation in preterms to be affected, giving rise to a high percentage of developmental defects in their primary dentitions (Stein, 1947; Grahnen & Larsson, 1958; Seow, 1986; Seow *et al*, 1987; Fearne *et al*, 1990).

1.2.2 Prevalence in preterm children

Table 1.1 lists the prevalence of enamel defects in studies of preterm low birthweight children, as previously reported in the dental literature, chronologically.

Early studies were done mainly on children with average birthweights of more than 2000g, for the reason that those with lower birthweights had little chance to survive. With the progress made on neonatal care in recent years, survival rates of infants born weighing less than 1500g have dramatically improved. More recent studies (Johnsen *et al*, 1984; Seow, 1986; Pimlott *et al*, 1985; Seow *et al*, 1987; Fearne *et al*, 1990) have included such children.

TABLE 1.1 PREVALENCE OF ENAMEL DEFECTS IN STUDIES OF PREMATURELY-BORN LOW BIRTHWEIGHT CHILDREN

AUTHORS/YEAR	PERCENTAGE (%) OF SUBJECTS WITH DEMONSTRABLE ENAMEL DEFECTS (Number of Subjects Examined)		
	LBW*	VLBW**	NORMAL
Stein, 1936	42 (12)		
Stein, 1947	50 (16)		
Forrester & Miller, 1955	20 (34)		
Kreshover, 1958	77 (35)		
Grahnen & Larsson, 1958	21 (68)		2 (61)
Rosenzweig & Sahar, 1962	24 (21)		1 (80)
Grahnen *et al*, 1974	43 (82)		15 (39)
Rosenstein, 1974	45 (62)		
Funakoshi *et al*, 1981	27 (52)		
Mellander *et al*, 1982	30 (91)		40 (48)
Johnsen *et al*, 1984		52 (67)	26 (46)
Pimlott *et al*, 1985		38 (87)	
Seow, 1986		79 (63)	
Seow *et al*, 1987	27 (33)	62 (77)	12 (47)
Fearne *et al*, 1990	70 (50)	83 (60)	37 (93)

* LBW: Low birthweight (1500g to less than 2500g)

** VLBW: Very low birthweight (less than 1500g)

Table 1.1 above was modified from Seow (1992)

The prevalence of enamel defects in the low birthweight preterm children ranged from between 21 to 77% and in the very-low birthweight preterm children the reported figures ranged from between 38 to 83%. A trend exists in nearly all reported studies. In each study the percentages of enamel defects in the low birthweight (LBW) preterm children is higher than that of full-term children and the percentage reported for the very low birthweight (VLBW) preterm children is even higher than in the low birthweight (LBW) preterm children.The following provides a brief summary of each of the listed studies:

As early as 1936, Stein has noted enamel defects in five out of the twelve preterm children he examined (Stein, 1936). He further substantiated his findings by demonstrating 50% of his later sample of sixteen preterm children had enamel hypoplasia located in the incisal third of their deciduous teeth (Stein, 1947).The children in his study were mostly born in the early part of the third trimester with birthweights of more than 2000g.

Following Stein (1947), Forrester and Miller (1955) documented severe enamel defects in the primary dentition of seven (20%) of the thirty-four preterm children they sampled. Kreshover *et al* (1958) conducted histological examinations and observed enamel defects in twenty-seven (77%) of the thirty-five premature non-surviving subjects.

In the same year, Grahnen and Larsson (1958) demonstrated enamel defects in fourteen (21%) of the sixty-eight preterm children (birthweights less than 2500g) compared to only one (2%) of the sixty-one full-term control sample (birthweights more than 3000g). This was later verified by

Rosenzweig and Sahar (1962) who showed five (24%) of the twenty-one preterm group (birthweight less than 2300g) had enamel defects compared to one (1%) of the eighty children of the control group (birthweight more than 2300g).

Grahnen *et al* (1974) found eighteen (22%) of the eighty-two preterm children had enamel hypoplasia with only two (5%) of the thirty-nine controls. They further found seventeen (21%) preterm children had enamel opacities with four (10%) in controls. These results by Grahnen *et al* (1974) gave a total of thirty-five (43%) preterm children with enamel defects compared to six (15%) controls.

Funakoshi *et al* (1981) found enamel defects in fourteen (27%) of the fifty-two preterm children investigated.

Mellander *et al* (1982) reported twenty-seven (30%) preterm children with enamel defects compared to nineteen (40%) in controls. The study by Johnsen *et al* (1984) who included preterm children with very low birthweights (less than 1500g) showed fourteen (21%) had enamel hypoplasia, twenty-one (31%) had opacities thus resulting in a total of thirty-five (52%) with enamel defects.

Pimlott *et al* (1985) noted maxillary primary incisors with enamel defects in thirty-three (38%) of their eighty-seven very low birthweight (VLBW) sample.

Seow (1986) found enamel defects in fifty (79%) of her sixty-three VLBW sample. Of the fifty (79%) with enamel defects, fifteen (24%) had enamel opacities alone. Seow and her co-workers (1987) further substantiated

her findings with a controlled study showing forty-eight (62%) of the very low birthweight, (VLBW, less than 1500g) children with enamel defects, compared to nine (27%) of the low birthweight (LBW, 1500-2500g) and six (13%) of the normal birthweight group. This study also demonstrated that the VLBW group are more susceptible than the LBW group to enamel defects with the control group being least affected.

Fearne *et al* (1990) reported similar findings with fifty (83%) VLBW children experiencing enamel defects compared to thirty-five (70%) in the LBW group and thirty-four (37%) in the control group.

1.2.3 Aetiological factors of enamel defects

Seow *et al* (1984a, 1984b) first proposed a broad classification for enamel defects (i.e. generalised or localised defects) in preterm children based on their aetiological origins.

Generalised defects are those usually found symmetrically distributed and are usually associated with systemic illnesses (Johnsen *et al*, 1984; Seow *et al*, 1984a); whereas localised defects are related to local trauma on the alveolus.

1.2.3.1 Generalised defects

With the higher prevalence of enamel defects found in preterm children, attempts have been made by several investigators to identify causative perinatal and neonatal systemic factors but most have been unconvincing. Investigations considering individual factors in isolation (Grahnen *et al*, 1969; Mellander *et al*, 1982) without taking into consideration many other systemic medical conditions that occur concurrently have failed,

as these variables occurring simultaneously are not controlled. Also in children who have enamel defects, most if not all important systemic causative factors are found, making it impossible to determine the relative importance of each individual causative factor.

Factors investigated included anaemia, hyperbilirubinaemia, respiratory distress syndrome, rickets, hypothermia, hypocalcaemia, nutrition, and Apgar scores (Funakoshi *et al*, 1981; Johnsen *et al*, 1984; Pimlott *et al*, 1985).

Due to the many difficulties encountered in determining the relative importance of individual systemic conditions in the pathogenesis of enamel hypoplasia, Seow *et al* (1989) examined a central mechanism upon which many of the systemic factors may operate in preterm children. The central mechanism being osteopenia or mineral deficiency. This has also been described as metabolic bone disease of prematurity (Brooke and Lucas, 1985).

The relationship between osteopenia and enamel defects was first demonstrated by Seow *et al* (1989) in a controlled study where all prematurely-born children with enamel defects had greater degrees of osteopenia (as measured by the neonatal radiographical cortical thickness of the humerus bone), compared to children without the enamel defects.

Mineral deficiency may provide a central mechanism through which other systemic factors act and is supported by evidence that respiratory distress syndrome, infections, hyperbilirubinaemia, anaemia, as well as

intracranial haemorrhage, are all associated with radiological and other evidence of decreased mineral stores (Koo *et al*, 1982; Greer & Tsang, 1985).

Low serum calcium (i.e. hypocalcaemia) as proposed by Nikiforuk and Fraser (1981) as being responsible for enamel defects has not been supported by medical findings. Serum levels of calcium in the neonatal period of infants remained fairly constant even in cases of extreme calcium deficiency (Masel *et al*, 1982; Binstadt & L'Heureux, 1978).

Although hypocalcaemia is unlikely to play a major direct role in the pathogenesis of enamel defects, it may be hypothesised that as mineral stores are depleted in an infant to maintain serum homeostasis, calcium and phosphate are also prevented from entering dental structures perhaps through homeostatic mechanisms mediated via the parathyroid hormonal axis (Seow, 1992).

The existence of a central mechanism of damage to developing enamel does not however, exclude the possibility that individual systemic factors may also work through other known mechanisms. For example, direct cellular damage to the ameloblasts by infective agents in the case of enamel hypoplasia caused by infection (Seow, 1992).

1.2.3.2 Localised defects

In the preterm children defects due to local trauma to developing primary teeth may be the results of either laryngoscope and/or endotracheal intubation:

1.2.3.2.1 Laryngoscopy

Children who were previously intubated and mechanically ventilated tended to suffer more enamel defects on the left maxillary anterior teeth (Illustrations 1.0 & 1.1) compared to non-intubated children (Seow *et al*, 1987). These defects were observed in the VLBW children but not in the LBW or normal children as they were not usually intubated.

The enamel defects localised to the left side of the mouth, may be explained by the position of the laryngoscope blade in contact with the alveolar mucosa during laryngoscopy. During this procedure the laryngoscope is inserted centrally into the mouth, but is then moved to the left of the midline in order to insert the orotracheal tube in the groove along the right side of the laryngoscope regardless of whether the operator is right of left-handed (Brooks, 1982).

In very small infants, particularly those of VLBW, the mandible is so hypoplastic that it does not allow an adequate pivot for the lifting of the anterior oropharynx and tongue to expose the laryngeal opening. Inadvertently, force is applied onto the left maxillary anterior alveolar ridge where the blade contacts, probably disrupting dental development in this region.

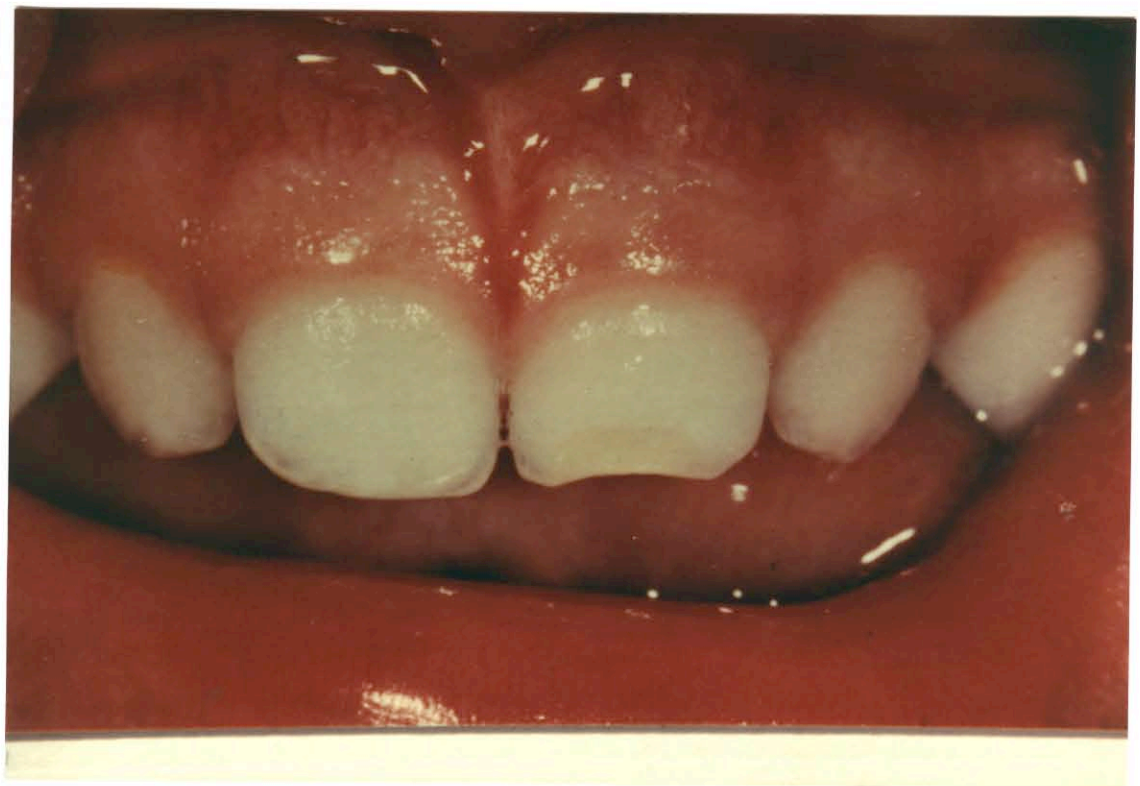


Illustration 1.0 Upper left maxillary central incisor affected by enamel defect on the incisal third of crown (Courtesy of Associate Professor K Seow)

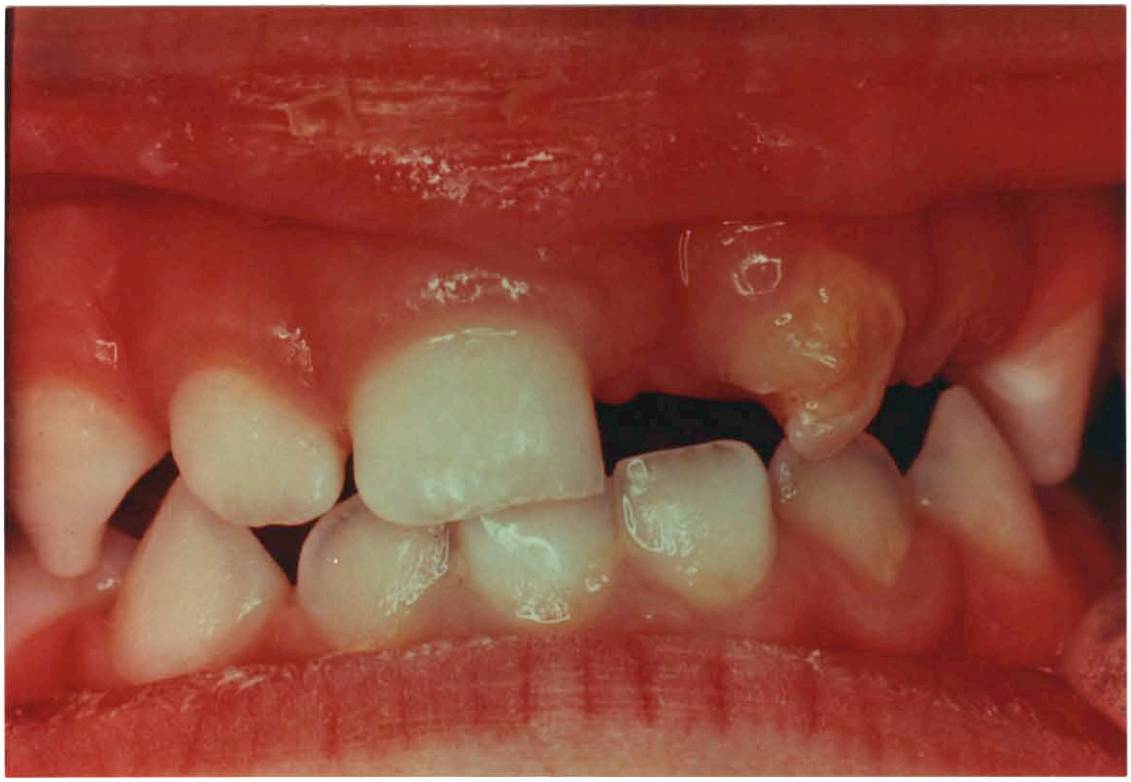


Illustration 1.1 Severe disruption of crown and root formation, as well as enamel defect located on the upper left incisors

Furthermore, general systemic mineral deficiency causing the thinning of bony cortices may lead to the developing teeth lying within the alveolar bone being easily injured through external traumatic forces, thus resulting in enamel hypoplasia. In support of this, Seow *et al* (1989) noted that the prematurely born children most susceptible to the traumatic effects of laryngoscopy are also those who showed the highest level of osteopenia.

1.2.3.2.2 Orotracheal tube

Indentations of the maxillary alveolar ridges have been noted where orotracheal tubes had been placed in LBW infants and examination of the developing incisors of these areas at autopsy had shown crown dilacerations with instances of tooth bud necrosis (Boice *et al*, 1976; Wetzel, 1980; Krous, 1980).

The above observation is not supported by the study of Seow *et al* (1987) where they suggested the pressure from the orotracheal tube was evenly distributed and not localised to the one side of the jaw, as the infants were routinely turned to lie alternately on both sides so that any pressure resulting from the orotracheal tube would be evenly distributed (Illustration 1.2). Furthermore, the duration of intubation did not affect the number of defects found (Seow *et al*, 1984a).

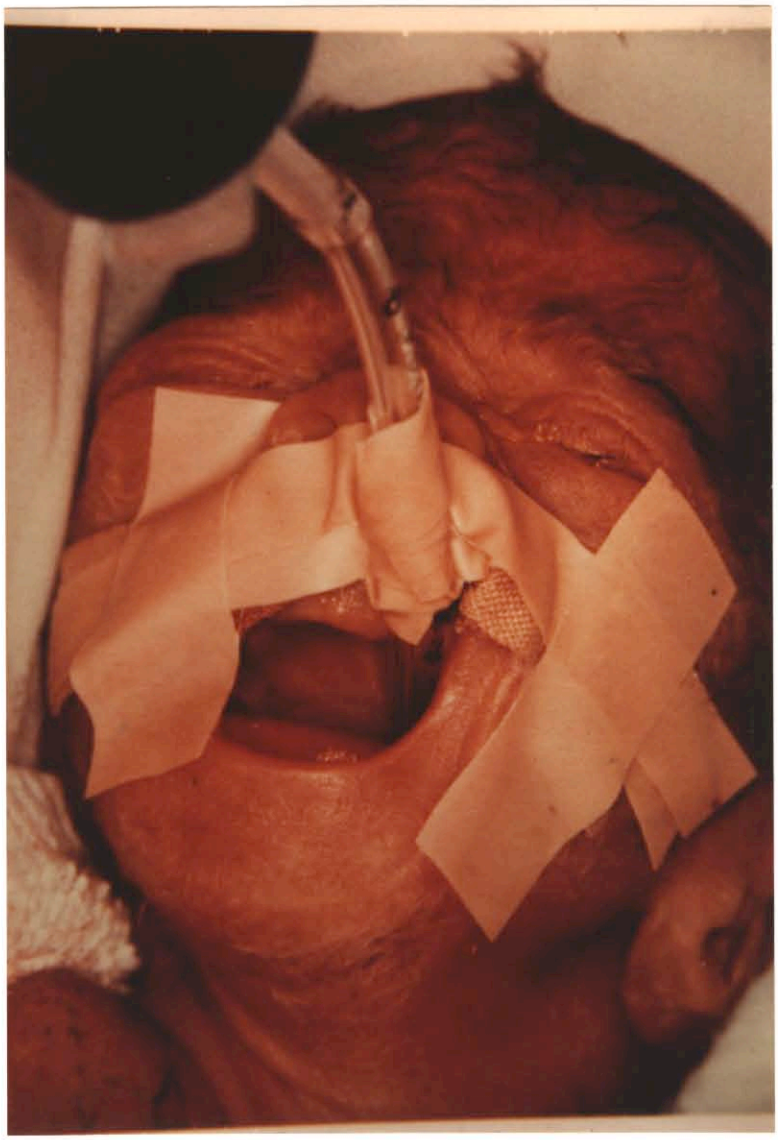


Illustration 1.2 A Preterm infant with orotracheal tube

1.3 Dental caries in preterm children

1.3.1 Prevalence

Studies on dental caries prevalence in the preterm children unfortunately are very few and far in between. The few available studies (Grahnen & Larsson, 1958; Rosenzweig & Sahar, 1962) on caries prevalence provide conflicting results.

According to the interpretation by Grahnen & Larsson (1958) of Stein's article published in the German literature in 1936, Stein was supposedly to have reported higher caries incidence and an increased tendency to tooth discolouration in the preterm children investigated. Stein (1947) in his review article summarising his previous works on enamel hypoplasia in preterm children, did not report on the caries prevalence in preterm children. Many other later investigators claimed that Stein (1947) in his paper supported higher caries susceptibility in preterm children. The confusion arose from the fact that Stein's paper in 1947 did report an increase caries susceptibility not in preterm but in full term children with chalky defects as a result of severe illness in the neonatal period.

Metabolic disturbances, severe illnesses or/and impairment of normal nutrition, occurring in early infancy of full term children can result in enamel lesions, not characteristically hypoplasia, but presenting as a chalky appearance, often stained, resembling mottled enamel without pits or depressions of the surface,without incisal edge involvement (i.e not associated with premature birth when hypoplasia is characteristically on the incisal edges). These chalky defects of calcification of the

postnatally formed enamel rather than defects in enamel matrix formation (hypoplasia) were observed to increase caries susceptibility (Stein, 1947).

Grahnen & Larsson (1958) were the first to publish data on the caries incidence of preterm children to include a control group as comparison. The caries incidence was assessed by the d.m.f.s.(decayed,missing,filled surfaces) system. No significant difference in caries incidence of the deciduous dentition could be found between the matched control and the preterm group at the average age of five years and four months. The d.m.f.s. of these thirty-five paired children were 16.8 for the preterm group and 16.3 for the control group (Grahnen & Larsson, 1958).

In 1962, Rosenzweig and Sahar who investigated preterm and full term children aged between 4 and 5 years, observed an average d.m.f.t.(decayed,missing,filled teeth) score of 4.38 in the twenty-one preterm children compared to an average d.m.f.t. score of 2.21 in the sixty-two full term children. Their results showed that caries was of a much higher prevalence in the preterm than in the controls. Therefore, the observation by Rosenzweig and Sahar (1962) was contradictory to that reported by Grahnen and Larsson (1958).

A recent study by Curzon *et al*, (1991) published only as an abstract, reported dental caries in two out of twenty-eight preterm children (d.m.f.t. of 4.0 and 11.0) as compared with only one out of twenty-one full term (d.m.f.t. of 2.0). These children examined had mean ages of 2 years 11 months for the preterm and 2 years 7 months for the full term controls. Higher d.m.f.t. scores in the preterm may lend some support to

the view of increased caries susceptibility in the preterm, but the small number of caries affected preterms and controls make any conclusions difficult.

1.3.2 Aetiology of dental caries in preterm children

While the major aetiological factors in dental caries are well established as microbial (in particular *Streptococcus mutans*), substrate (sugar) and host (eg: susceptible tooth surfaces), the relative importance of these and other subsidiary factors have not been well established.

There has been a great surge of interest recently, looking at the risk of various factors to caries. Large scale studies in children have been undertaken (Leverett *et al*, 1993a; Leverett *et al*, 1993b; Beck *et al*, 1992; Graves *et al*, 1992; Disney *et al*, 1992; Graves *et al*, 1991). These studies had attempted to assess the relative contribution of known aetiological factors. Being given the name - Caries risk assessment studies - their investigations have mainly concentrated on the general paediatric population with little attention to subgroups (for example the preterm children) whose teeth may have inherent susceptibility (a host factor) to caries. Paucity of information exists as to how factors such as enamel defects, fluoride intake, Streptococcus mutans levels, dietary oral hygiene and other behavioural habits with time contribute towards dental caries in preterm children. These variables will be scrutinised in our caries risk assessment analyses of the subjects studied.

1.4 Relationship between enamel defects and dental caries

Despite numerous studies on premature infants reporting on the prevalence of enamel defects and possible causative factors, follow-up longitudinal studies to investigate the sequelae of these enamel defects are not available. Do these defects eventually become carious lesions? Few researchers have attempted at answering this question.

The relationship between caries and enamel hypoplasia in preterm children had previously been reported in only two studies (as mentioned in section 1.3.1), and these studies were cross-sectional in nature. Also, while these studies have reported on both the prevalences of enamel defects and dental caries, it was unclear as to whether the caries occurred on the same teeth or surfaces involved in enamel defects.

Rosenzweig and Sahar (1962) reported that caries prevalence was higher in the preterm group and it mainly affected preterm children manifesting enamel hypoplasia. The average d.m.f.t. was 12.2 for five preterm children with enamel hypoplasia compared to a d.m.f.t. score of 1.9 for the remaining sixteen preterm children without hypoplasia. Caries prevalence of preterm children without hypoplasia was <u>not</u> significantly different from full-term controls (Rosenzweig & Sahar, 1962).

The relationship of enamel defects to dental caries in population groups other than prematurely born children has been looked at in many cross-sectional studies. However, these studies again had the same deficiency as the others in that the coincidence of occurrence of both decay and enamel defects was not established. (McCall & Krasnow, 1938; Davies,

1939; Allen, 1941; Bibby, 1943; Staz, 1944; Carr, 1953; Rosenzweig & Sahar, 1962, Baume & Meyer, 1966; Infante & Gillespie, 1974; Infante *et al*, 1975; Rodda & Smillie, 1984; Nikiforuk & Fraser, 1984). Prehistoric population groups were also studied (Cook & Buikstra, 1979; Williams & Curzon, 1986; Duray, 1990).

In summary, the results reported by the above investigators were thus unclear. It remains to be determined as to whether enamel defects render teeth more susceptible to caries or whether enamel defects reduced the susceptibility of teeth to caries. The conflicting results obtained from previous reports were also partly due to the confusion as well as lack of classification of enamel defects. Hypoplasia as a term, was used to encompass all developmental enamel defects in many early studies (McCall & Krasnow, 1938; Davies, 1939; Allen, 1941; Bibby, 1943; Staz, 1944; Carr, 1953; Rosenzweig & Sahar, 1962). Enamel defects due to undermineralisation (hypocalcification or opacities) were not separated from those due to deficiency in matrix formation (hypoplasia). It is now generally accepted that these two types of enamel defects are clinically distinct. Enamel hypoplasia (Illustration 1.3) is usually defined to mean a quantitative defect with reduced thickness of the enamel surface and may be manifested as pits, grooves or missing enamel (FDI, 1982). In contrast, an opacity (Illustration 1.4) is usually diagnosed as a change in translucency without loss of quantity of enamel.

Concern over the need to identify the different types of enamel defects and the use of descriptive categories of defects independently by different authors (Baume & Meyer, 1966; Infante & Gillespie, 1974; Infante *et al*, 1975; Rodda & Smillie, 1984) had led to the development

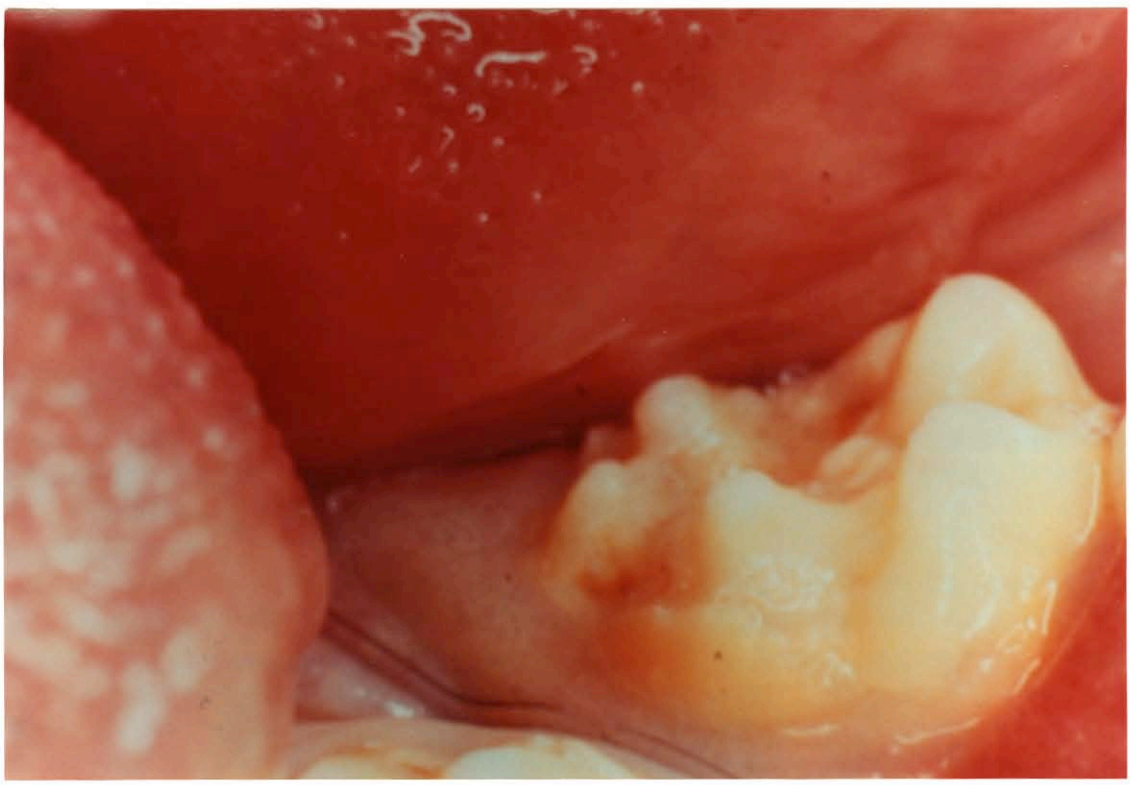


Illustration 1.3 Enamel hypoplasia located on the deciduous lower second molar

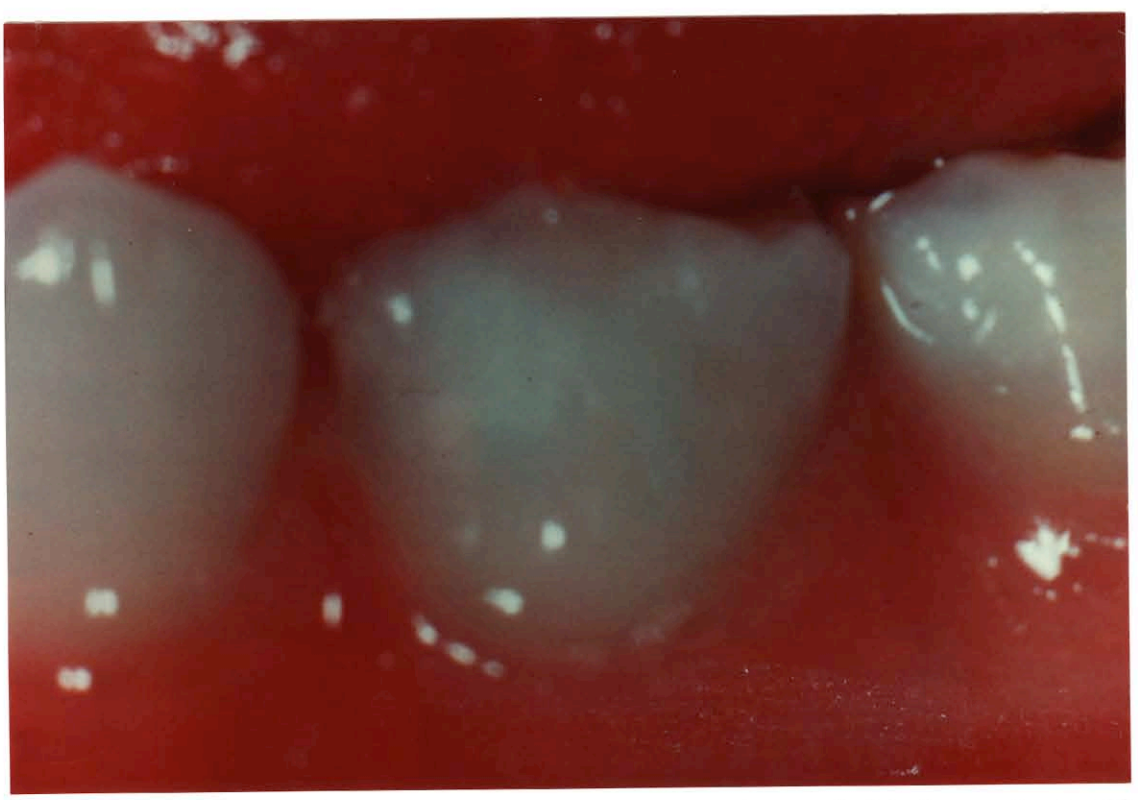


Illustration 1.4 Enamel opacity located on labial surface of tooth 74

of a standardised terminology and internationally accepted classification system - the Developmental Defects of Enamel (D.D.E.) index in 1982, by the Federation Dentaire International (FDI). Clarkson (1989) later modified the D.D.E. index to reduce the number of scores required for each defect.

This present study aims to indentify enamel defects according to the modified D.D.E. index and investigate the sequelae of these enamel defects, making use of follow-up examinations over a period of two years.

CHAPTER 2 PATIENTS AND METHODS

2.1 PATIENTS

2.1.1 Preterm children

Children who were born prematurely with birthweight of less than 1500g were observed to experience the highest prevalence of enamel defects compared to greater birthweight preterm groups (Seow *et al*, 1987). The very low birthweight (VLBW, less than 1500g) group having the greatest number of enamel defects was therefore selected as the group to study in order to determine the outcome of these enamel defects.

Children selected for this present study were recruited from the Mater Public Hospital, South Brisbane. This hospital is one of the two major centres for the retrieval of prematurely born babies in Queensland. The children in the VLBW (less than 1500g) sample were those born in the period 1989-1992 who were attending the Growth and Development Clinic of the hospital. This clinic first set-up in 1978 aims to provide multi-disciplinary follow-up management of all surviving infants with low birthweights. VLBW infants are considered to be at high risk for developmental problems. With adequate follow-up, early diagnosis and intervention can be made which eventually leads to better long term prognosis. The other major benefit of establishing this clinic is that an assessment of perinatal factors that may adversely effect outcome can be made, and methods of perinatal intensive care thought to have longitudinal adverse effect can be improved upon.

Patients recruited from the Growth and Development Clinic are screened regularly for various areas of medical concerns. However, follow-up dental assessment has not been attempted prior to this present dental study.

2.1.2 Control children

Healthy full term children in the normal birthweight group (more than 2500g) serving as controls were selected at random from the birth register at the same hospital. These children were matched for age, sex and race of the preterm subjects.

Explanatory letters and consent forms (Appendix I-V) were sent out to parents of both VLBW and control samples requesting their participation in the study that required three dental examinations.

2.2 Medical and Dental Histories

On presentation to the Paedodontic Clinic of the University of Queensland Dental School, postnatal medical and dental histories were taken of each of the participating children (Appendix VI). In addition, maternal medical history during pregnancy and neonatal history of each child were also retrieved from hospital records. Relevant personal details of parents including occupation(s), maternal age, and other demographic information were also obtained (Appendix VII).

The socioeconomic status (S.E.S.) of each participating child was established based primarily on the occupation of the head of the household. If the occupation(s) of the parent(s) were not stated, the suburb of residence was used to determine the S.E.S. as outlined in the Australian Census (1991). S.E.S. has been reported to be related to caries experience in siblings (Disney *et al*, 1992, Bohannen *et al*, 1985

and Abernathy *et al*, 1987). Participants were classified into the following four categories based on their socioeconomic standings shown below (modified from Harth & Thong, 1990; Powell & McEniery, 1987):

I (High) : Professional, Administrative, Executive, Managerial, Technical and related workers

II (Middle): Clerical and sales

III (Low) : Labourers, tradesmen, farmers and related workers

IV (Others): Home duties, students, unemployed, retired and others.

Dental histories also included previous dental treatment, fluoride supplementation, trauma to teeth, and oral hygiene habits(Appendix VI).

2.3 Dietary History

At each dental visit, a dietary evaluation form (Appendix VIII) was issued for each participants with a stamped, self-addressed envelope in order to be conveniently returned to the author. Their parents were requested to fill in a detailed account of what food items had been consumed during and in between meal times for three consecutive days (including one weekend day). The dietary form was then analysed tc determine the mean daily sugar frequency, and form of sugar consumed.

2.4 Dental Examination

Clinical dental examinations were performed at the Paedodontic Clinic of the University of Queensland Dental School in a standardised manner using a dental mirror and a probe (Ash no. 54) under normal dental lighting.

Extra-oral and intra-oral abnormalities were recorded in comprehensive examination forms (Appendix IX). The teeth present, type of occlusion, and the degree of anterior overbite and overjet were first noted (Appendix X).

The teeth were then dried and each tooth surface examined for the presence of dental caries and developmental defects. Dental caries were recorded using World Health Organisation (WHO) criteria (WHO, 1987). In the WHO (1987) caries charting system, the lesions were classified either as cavitation or intact. The early (white spot) lesions were not recorded as carious.

Enamel defects (opacities and hypoplasia) were classified according to the modified Developmental Defects of Enamel (D.D.E.) Index (Clarkson, 1989). Table 2.1 provides the definition of various types of enamel defects. The modified D.D.E. Index from Clarkson (1989) is shown in Table 2.2.

TABLE 2.1 Definition of various types of enamel defects.

DEFINITION OF VARIOUS TYPES OF ENAMEL DEFECTS (FDI, 1982)		
Opacity	–	qualitative defect in enamel, abnormality in translucency of enamel
Hypoplasia	–	quantitative defect in enamel, reduced thickness of enamel
Discoloured enamel	–	abnormal appearance in enamel
Developmental defects of enamel	–	disturbances in hard tissue matrices and their mineralization during odontogenesis

TABLE 2.2 The modified D.D.E. Index from Clarkson (1989).

MODIFIED DDE INDEX FOR USE IN GENERAL PURPOSE EPIDEMIOLOGICAL STUDIES

Categories	Code
Normal	0
Demarcated opacities	
white/cream	1
yellow/brown	2
Diffuse opacities	
" – Lines	3
" – Patchy	4
" – Confluent	5
Confluent/patchy + staining + loss of enamel	6
Hypoplasia	
Pits	7
Missing enamel	8
Any other defect	9
Extent of Defect	
Normal	0
<⅓	1
at least ⅓ <⅔	2
at least ⅔	3

Agenesis and irregularities of crown form of the teeth were also recorded on the examination form (Appendix IX).

Oral hygiene indices were computed based on a modified plaque index of Silness-Löe as advocated by Ainamo and Bay (1975). A standard sickle probe (Ash no.54) was used to detect the presence of supragingival plaque without trauma to the gingival tissue. A score (0 for absence, 1 for presence) was recorded on each of the three surfaces (mesial, buccal and lingual) of six individual teeth (55, 61, 64, 75, 81, 84 - numbers indicate FDI System [1971], of tooth identification) (Appendix IX).

Bitewing radiographs were taken only when indicated to assist caries diagnosis and with parental consent. Panoramic radiographs were obtained in individuals where abnormalities were suspected.

On completion of the first examination, the parent(s) of the child concerned were informed of the findings and treatment needs, if any. Restorative and preventive dental care were offered to children who did not have their own dentists or relevant records were forwarded to their respective dentists.

2.5 Follow-up dental examinations

Follow-up examinations were performed at one and two years after the initial visit. Examinations were performed without reference to previous dental charting of each child to prevent examiner bias.

2.6 *Streptococcus mutans* level

Commercially available dip-slide system testing kit for *Streptococcus mutans*, Dentocult SM (Orion Diagnostica, Finland) was employed as a microbiologic indicator of the level of *Streptococcus mutans* infections (Illustration 2.1).

The technique of *Streptococcus mutans* (*Strep. mutans*) collection entailed sliding the test strip gently over the dorsal surface of the tongue alternating between the two surfaces of the test strip ten times in total. The test strip was then immediately immersed into the culture broth solution in the vial supplemented with a bacitracin-impregnated disc left in tube for at least fifteen minutes prior to making use of test strip in patient. The vial was recapped and remained one quarter turn open. The culture vial was placed in an incubator set at 35°C and incubated for 48 hours.

When more than one test strip is to be done, the bacitracin discs can be added to the vials beforehand. However, each vial can only hold one test strip, and the vials must be used during the same day. The bacitracin disc allow the broth medium to selectively promote the growth of *Strep. mutans* only.

Illustration 2.2 shows three selected vials with different concentrations *Strep. mutans* colonies on the three test strips following incubation.

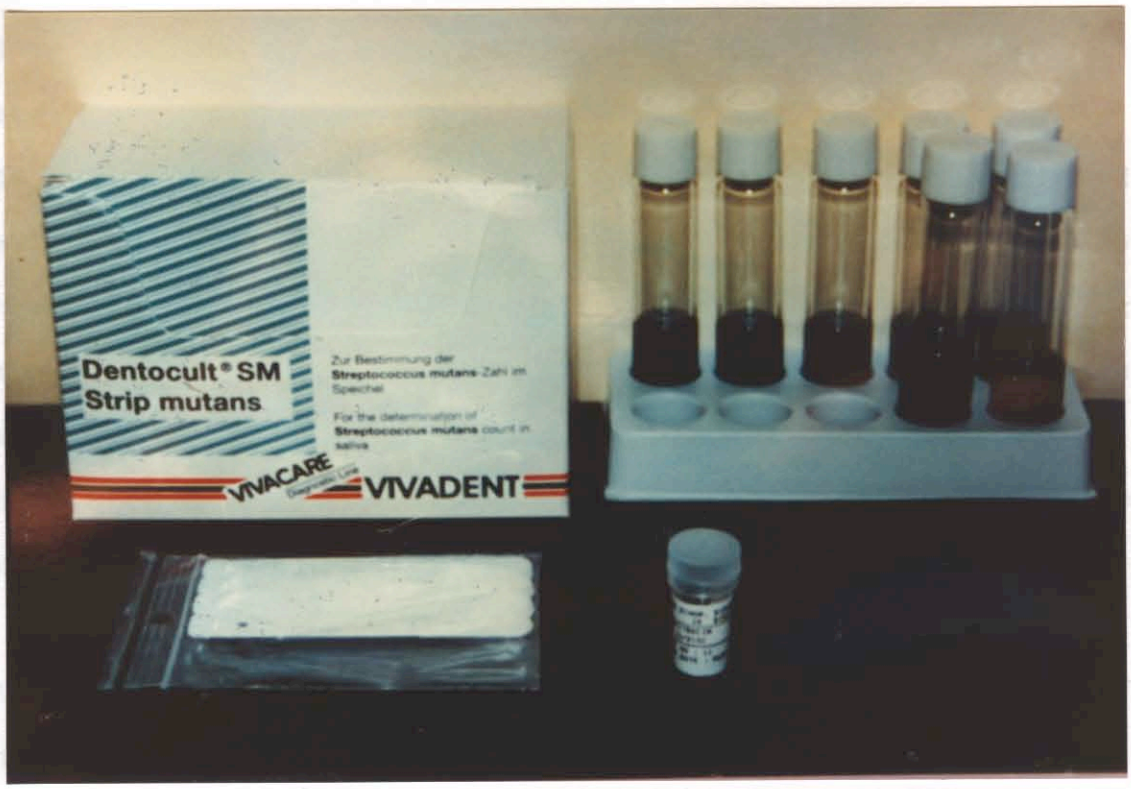


Figure 2.1 Commercially available dip-slide system testing kit for *Strep. mutans*

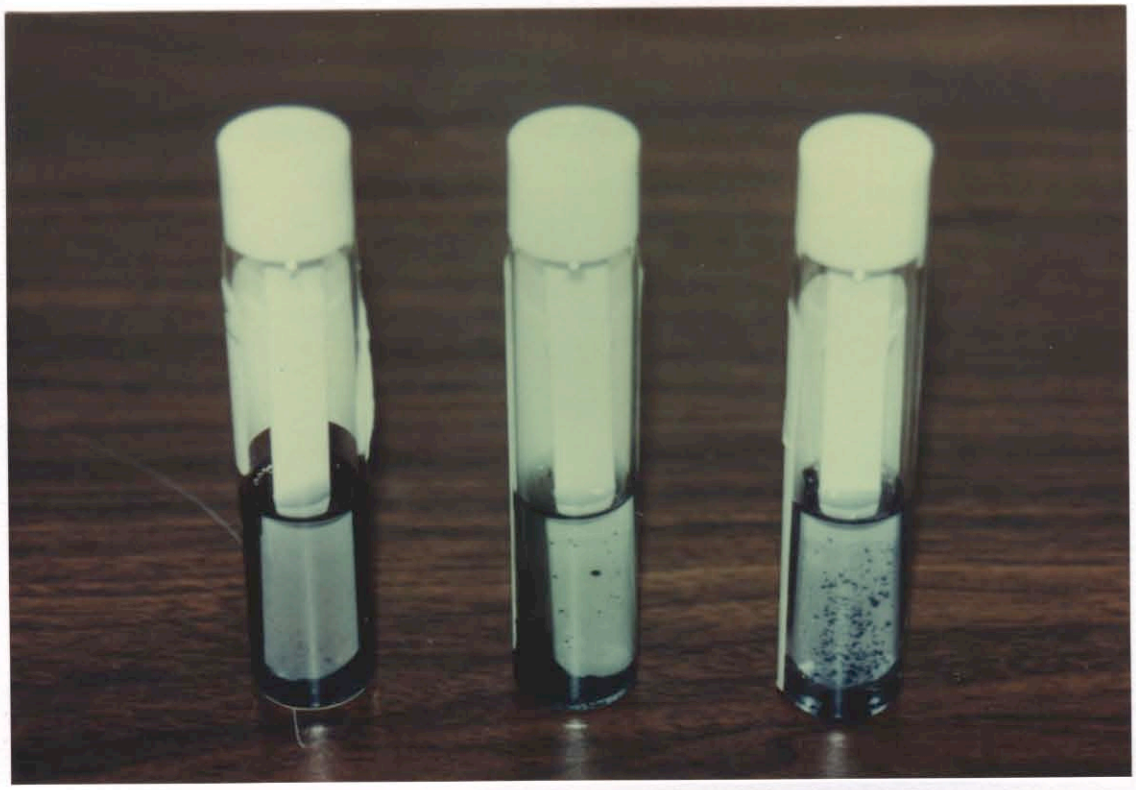


Figure 2.2 Three selected vials with different concentrations *Strep. mutans* colonies on test strips, following incubation

Evaluation of *Strep. mutans* levels according to the instructions from the manufacturer are as follow:

The *Strep. mutans* bacteria in the specimen will adhere to the treated side of the strip in proportion to their actual number in the saliva. The number of *Strep. mutans* bacteria per ml saliva is obtained by comparing the test strip with evaluation chart (Fig. 2.3) supplied by the manufacturer.

Fig. 2.4 shows four test strips corresponding to that of the evaluation chart.

The corresponding *Strep. mutans* levels in the saliva for Classes 0-3 of the above evaluation chart

Class 0 & 1 : < 100,000 bacteria/ml saliva

Class 2 : corresponds to a count between Class 1 and Class 3

Class 3 : >1,000,000 bacteria/ml saliva

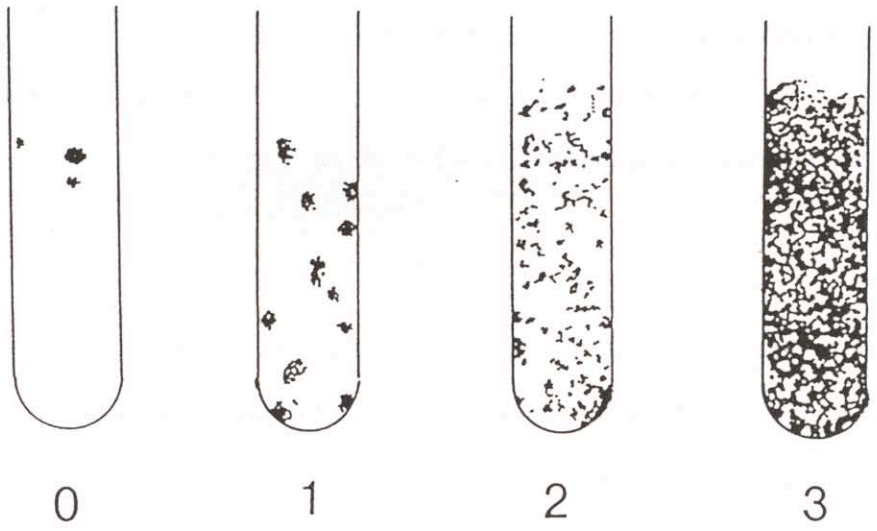


Illustration 2.3 Evaluation Chart for the treated side of incubated test strips

Illustration 2.4 Test strips corresponding to classes on the evaluation chart

2.7 Intra-examiner reliability

Dental examinations on both control and study groups were carried out by the one examiner thus negating inter-examiner inconsistency. This is likely to improve reliability with reduced sources of methodological error.

Intra-examiner reliability was assessed using kappa statistic, which corrected the observed proportion of agreement for the proportion of agreement to be expected by chance alone. The statistic, kappa is a useful descriptor of intra-examiner consistency (Fleiss *et al*, 1979). Mathematically, kappa (K) is represented by the formula

$$K = \frac{P_o - P_e}{1 - P_e} = \frac{\sum_{i=1}^{N}\sum_{j=1}^{k} n_{ij}^2 - Nn\left(1 + (n-1)\sum_{j=1}^{k} p_j^2\right)}{Nn(n-1)\left(1 - \sum_{j=1}^{k} p_j^2\right)}.$$

where N is the number of subjects on a categorical variable.

n is the number times the subjects were repeatedly examined.

k is the number of possible categories of a variable.

n_{ij} denoting the number of judgements on subject i (i=1,...,N) which were in category j (j=1,...,k).

Σ is the Greek letter capital sigma to indicate "summation of" with upper and lower limits of summation.

P_o is the observed proportion of agreement.

P_j is the overall proportion of judgments in category j

P_e is the expected proportion of agreement

In order to apply the statistical test, kappa, five participants in the VLBW group generally experiencing higher prevalence of enamel defects were examined twice at six weeks apart.

In interpreting values of *K*, for most purposes, then, values below 0.4 may be taken to represent poor or only fair reliability, values between 0.41 and 0.60, moderate reliability, values between 0.61 and 0.80, substantial reliability, and values between 0.81 and 1.00 almost perfect reliability (Landis and Koch 1977). It is to be noted that this statistical test is appropriate when prevalence rates are approximately 50% and collapses when prevalence rates are zero or 100%, regardless of the reliability of method (Anderson *et al*, 1993).

2.8 Statistics

2.8.1 Statistical tests and level of significance used

The Student's *t*-test (unpaired), Fisher's Exact test (for 2x2 tables) and Chi-square test where indicated, were applied for statistical analyses of the data. P-values quoted were two tailed.

The probability level of significance used in the processing and reporting of data was at the 0.05 level unless otherwise stated.

CHAPTER 3 RESULTS

3.1 Consent rates

3.1.1 Preterm children

Fifty-five letters were sent requesting parents of preterm children to participate in this study. These children were all known living VLBW preterms born at the Mater Public Hospital in the period May 1989- Dec 1992 and currently residing in different parts of Queensland. Of the fifty-five letters, eleven letters were returned with incorrect current addresses. Four parents refused participation, and two preterm children failed to attend in-spite of repeated rescheduling of appointment. The consent rate was therefore 64%. However, the participation rate was at 59% (Twenty-six preterm children participated). The main reason accounting for the artificially low participation rate was likely to be due to a high proportion of unreturned letters with incorrect addresses, and many (six out of eleven non-respondents) were residing in places very distant from Brisbane.

3.1.2 Control children

It was impossible to establish a consent rate for this group with any accuracy because addresses derived from hospital birth records, were approximately two years old. In all, one hundred and twenty-three letters were sent. Of these, twenty-four were eventually returned to the sender, with incorrect current addresses. It was not possible to determine as to how many of the seventy-three 'non-respondents' actually received the letters in the highly mobile young families in Brisbane. No parents refused participation. In total, we examined twenty-seven children (a participation rate of 27%).

3.2 Subject Attendance

3.2.1 Preterm group

Amongst the twenty-six preterm children participating , the number of these children not attending the first, second and third examinations were one(4%), seven(27%) and one child(4%) respectively. In other words, at the final examination twenty-five(96%) of the original study population attended, having been monitored for a period of two years on an average. Thus, only one preterm child(4%) dropped out of the study before its completion.

3.2.2 Control group

Amongst the twenty-seven(27) full-term control children participating, the number of these children not attending the first, second and third examinations were two(7%), eleven(59%) and seven(26%). This would mean that at the final examination, twenty(74%) control children attended with seven out of the twenty-seven children dropping out of the study before its completion.

3.3 Intra-examiner reliability: Kappa Statistic

Intra-examiner reliability was assessed using Kappa statistic (Fleiss *et al*, 1979). The Kappa values for intra-examiner reliability based on individual teeth examined are shown in Table 3.3.1. On the whole, the Kappa values indicated substantial reliability based on the commonly followed categorical scale by Landis and Koch, 1977 (Table 3.3.2). The judgements on the second primary molar and central incisor seem to be more consistent than those of other teeth with the lateral incisor being least consistent. On the average, all the observations have adequate intra-examiner reliability.

Table 3.3.1 **Mean values of Kappa for intra-examiner reliability on individual teeth**

	Maxillary		Mandibular	
	Right	Left	Right	Left
Second molar	1.000	1.000	0.730	1.000
First molar	0.429	0.566	0.678	0.524
Canine	0.714	0.444	0.655	1.000
Lateral incisor	0.412	0.718	-0.111*	0.565
Central incisor	1.000	1.000	1.000	1.000

Overall mean Kappa Value = 0.716±0.296

*This negative value signify a single judgement which was at variance with others.

Table 3.3.2 Kappa values on a categorical scale (Landis & Koch, 1977)

K < 0.40	represent poor or only fair reliability
0.41 < K < 0.60	represent moderate reliability
0.61 < K < 0.80	represent substantial reliability
0.81 < K < 1.00	represent almost perfect reliability

3.4 Demography of participating children

In all a total of, fifty-three of the children participated in the study. The mean (± SD) ages of all the children in the study at the initial examination was 29.8 ± 4.5 months (range 23 - 38 months) and at the end of the final examination was 52.2 ± 4.7 months (range 44 - 61 months).

The VLBW group comprised of twenty-six children (12 males, 14 females). The mean birthweight was 969 ± 243 g (range 652 - 1534 g) and mean gestation age was 27 ± 1.9 weeks (range 24 - 29 weeks). At Exam I, II and III the mean ages (months) ± SD of VLBW children were 27.4 ± 4.5, 41.3 ± 6.3, 51.9 ± 5.1 respectively.

In the normal birthweight matched control group, twenty-seven children (13 males, 14 females) were selected. These children were full term infants with a mean birthweight of 3396 ± 395g (range 2810 - 4110g) and mean gestational age of 39 ± 1.6 (range 37 - 42 wks). Children with prenatal, or perinatal and/or postnatal complications were excluded from this study. At Exam I, II and III the mean ages (months) ± SD of control children were 32.0 ± 3.1, 45.4 ± 3.8, 52.5 ± 4.2 respectively.

Table 3.4.1 provides a comparison of the number of preterms and controls studied, sexes of the participants, mean birthweights, mean gestational ages, socioeconomic standings and mean ages at each examinations. The major differences were found, as expected in the mean birthweights (969 g for the preterms, in contrast to 3,396 g for the controls)($P<0.0001$) and mean gestational ages (27 weeks for the preterms and 39 weeks for the controls)($P<0.0001$).

Table 3.4.1 Demographic data of subjects in study

	Preterms	Controls	P-value
Total Number	26	27	
Sex			
Male	12	13	N.S.
Female	14	14	N.S.
Mean Birthweight (g) ± SD	969 ± 243	3396 ± 395	P<0.0001
Range (g)	(652 - 1534)	(2810 - 4110)	
Mean Gestational Age (wks) ± SD	27 ± 1.9	39 ± 1.6	P<0.0001
Range (wks)	(24 - 29)	(37 - 42)	
Socioeconomic Status			
I (High)	12	9	
II (Middle)	3	2	
III (Low)	8	10	N.S.
IV (Others)	2	6	
Mean Age at Exam (month ± SD)			
I	27.4 ± 4.5	32.0 ± 3.1	N.A.
II	41.3 ± 6.3	45.4 ± 3.8	N.A.
III	51.9 ± 5.1	52.5 ± 4.2	N.S.

3.5 Prevalence of enamel defects

Table 3.5.1 summarises the prevalence of enamel defects in both the preterms and controls, together with their respective mean, standard deviation and range.

Enamel defects were found to affect preterm children more than control children in all three examinations. At Exam I, out of the total 25 preterm children presenting for examination, 22(88.0%) were affected by at least one enamel defect in contrast to only 10 of the 25 (40.0%) controls (P-value = 0.001). Similarly, at Exam II, 18 of the 19 (94.7%) preterm children presenting had enamel defects compared to only 6 of the 11 (54.5%) control children (P-value = 0.029). At the final examination (Exam III), 24 of the 25 (96.0%) preterms were observed to be affected by enamel defects in comparison to only 9 of the 20 (45.0%) controls (P-value = 0.0005).

Similar trends were observed in numbers of affected teeth (Table 3.5.1). At Exam I, 67 of the 377 (17.8%) teeth in the preterms were affected by enamel defects compared to only 16 of the 476 (3.4%) teeth in controls ($P < 0.00001$). At Exam II, 90 of the 392 (23.0%) preterm teeth were affected compared to 12 of the 220 (5.5%) teeth in control children ($P < 0.00001$). Similarly, at Exam III, 191 of the 499 (38.3%) teeth in preterm children were affected by enamel defects compared to 20 of the 399 (5.0%) teeth in the controls ($P < 0.00001$).

The mean number of affected teeth for each child with their standard deviations, SD are also shown in Table 3.5.1. At Exam I, the mean ±SD

Table 3.5.1 Prevalence of enamel defects in preterm and control children

	Exam I		Exam II		Exam III	
	Preterm	Control	Preterm	Control	Preterm	Control
Total no. of children	25	25	19	11	25	20
Total no. of teeth	377	476	392	220	499	399
Total Enamel Defects						
No. of affected children (%)	22(88.0%)	10(40.0%)	18(94.7%)	6(54.5%)	24(96.0%)	9(45.0%)
(P-value)·	0.001		0.029		0.0005	
No. of affected teeth (%)	67(17.8%)	16(3.4%)	90(23.0%)	12(5.5%)	191(38.3%)	20(5.0%)
(P-value)·	<0.00001		<0.00001		<0.00001	
Mean ± SD affected teeth/child	2.6±2.5	0.6±1.0	4.7±4.1	1.1±1.2	7.6±4.9	1.0±1.3
(P-value)··	0.0005		0.0086		<0.0001	
Range	0 - 12	0 - 4	0 - 17	0 - 3	0 - 16	0 - 4

(P-value)· from Chi-square, χ^2 test
(P-value)·· from Student's *t*-test (unpaired)

values for the preterm and control children were 2.6 ± 2.5 and 0.6 ± 1.0 respectively. At Exam II, the mean values for the preterm and control children were 4.7 ± 4.1 and 1.1 ± 1.2. Not surprisingly at Exam III, the mean values for the preterm and control children were 7.6 ± 4.9 and 1.0 ± 1.3 respectively.

The differences in the mean values between the preterms and controls were likewise extremely significant for Exam I (P-value = 0.0005) and Exam III (P-value < 0.0001), and very significant in Exam II (P-value = 0.0086).

The number of affected teeth for each child, at Exam I ranges from 0 - 12 for preterm children and 0 - 4 for controls. At Exam II the ranges were between 0- 17 and 0 - 3 for preterm and control children respectively. At the final exam, the ranges were from 0 - 16 and 0 - 4 for preterms and controls respectively.

It should be noted that many children may not have a complete primary dentition at Exam I and II. The higher prevalence of enamel defects observed in Exam III is due to the fact that the later erupting teeth (second molars) have the highest prevalence of enamel defects.

3.5.1 The number of preterm and control children affected with various types of enamel defects.

Figs.3.5.1 and 3.5.2 show the number of preterm and control children respectively, who were affected with the various types enamel defects at Exams I, II and III. These various types of enamel defects can be grouped into three types which are opacity (Illustration 3.1); hypoplasia (Illustration 3.2) and hypoplasia with opacity occurring concurrently (Illustration 3.3) (HO).

In the preterm group (Fig.3.5.1), enamel hypoplasia was seen most at Exam I, in 17 out of the 25 (68%) children. At subsequent exams, the number of preterm children affected with enamel hypoplasia were only slightly lower, that is 52.6% and 64.0% at Exam II and III respectively.

Enamel opacities affected preterm children most in Exam III (21 preterm children, 84.0%) with preterm children least affected by enamel opacities at Exam I (12 preterm children, 48%). At Exam II, 14 preterm children (73.7%) were affected by enamel opacities.

Enamel hypoplasia with opacities occurring concurrently (HO) on one or more teeth affected preterm children most in Exam III (21 preterm children, 84%). No children were found to have HO at Exam I. At Exam II, the author found 3 preterm children (15.8%) with HO.

In all, the total number of children seen with enamel defects as shown in Fig. 3.5.1 for Exam I, II and III were 22(88.0%), 19(94.7%), and 24(96.0%) respectively.

Fig.3.5.1 Percentage of Preterm Children with Enamel Defects

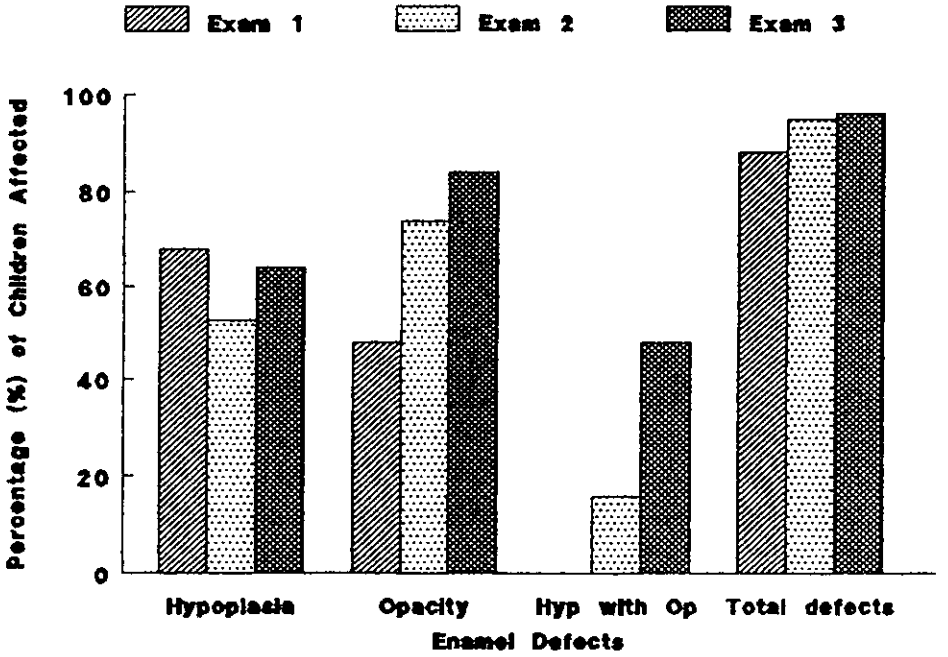
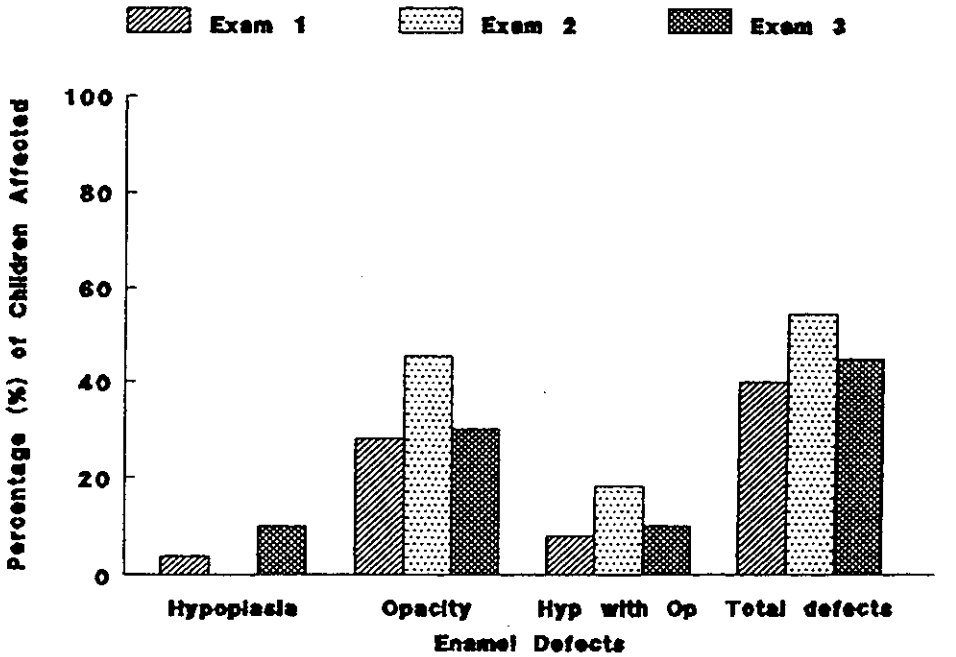


Fig.3.5.2 Percentage of Control Children with Enamel Defects



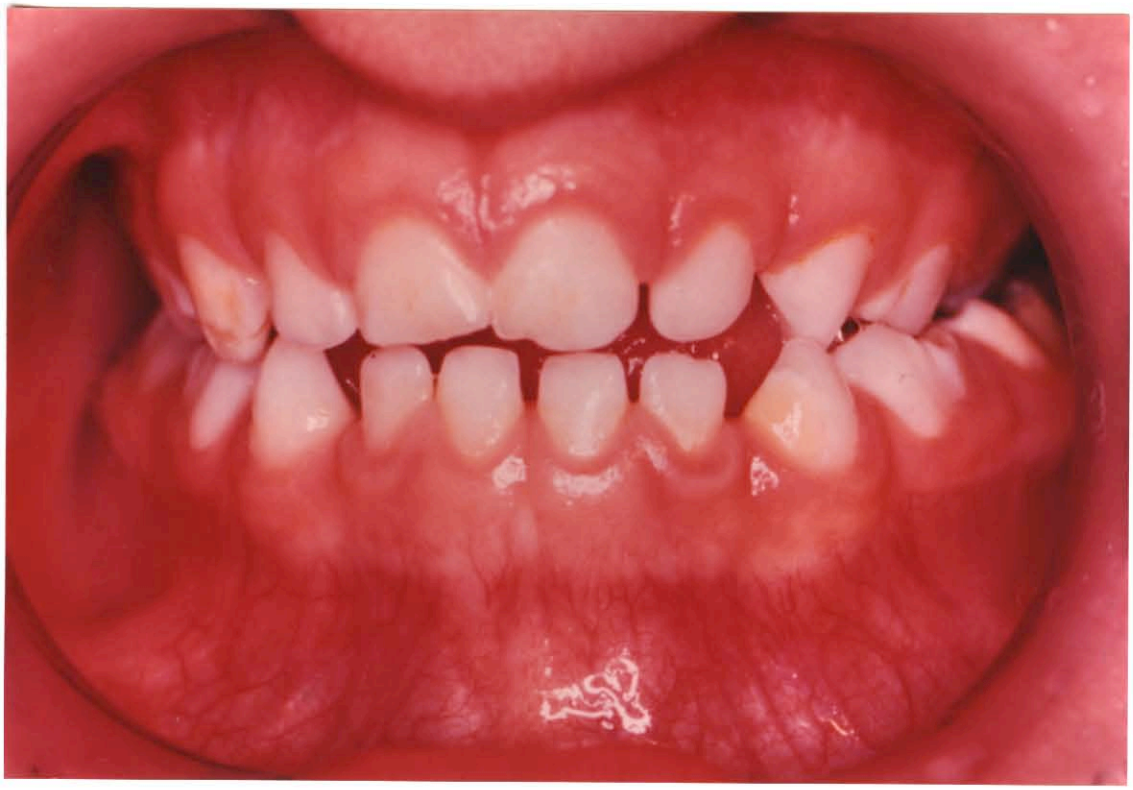


Illustration 3.1 White/Cream enamel opacity located on tooth 73

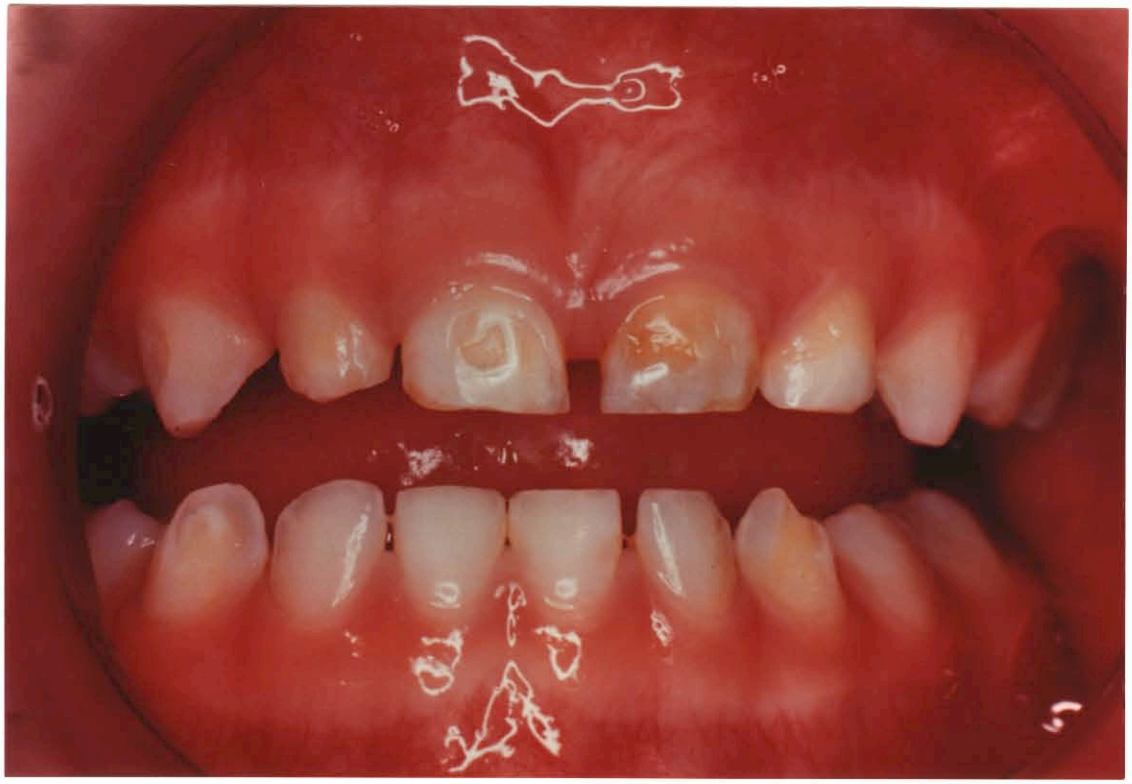


Illustration 3.2 Enamel hypoplasia located on labial surfaces of teeth 53, 51, 61, 73 and 83

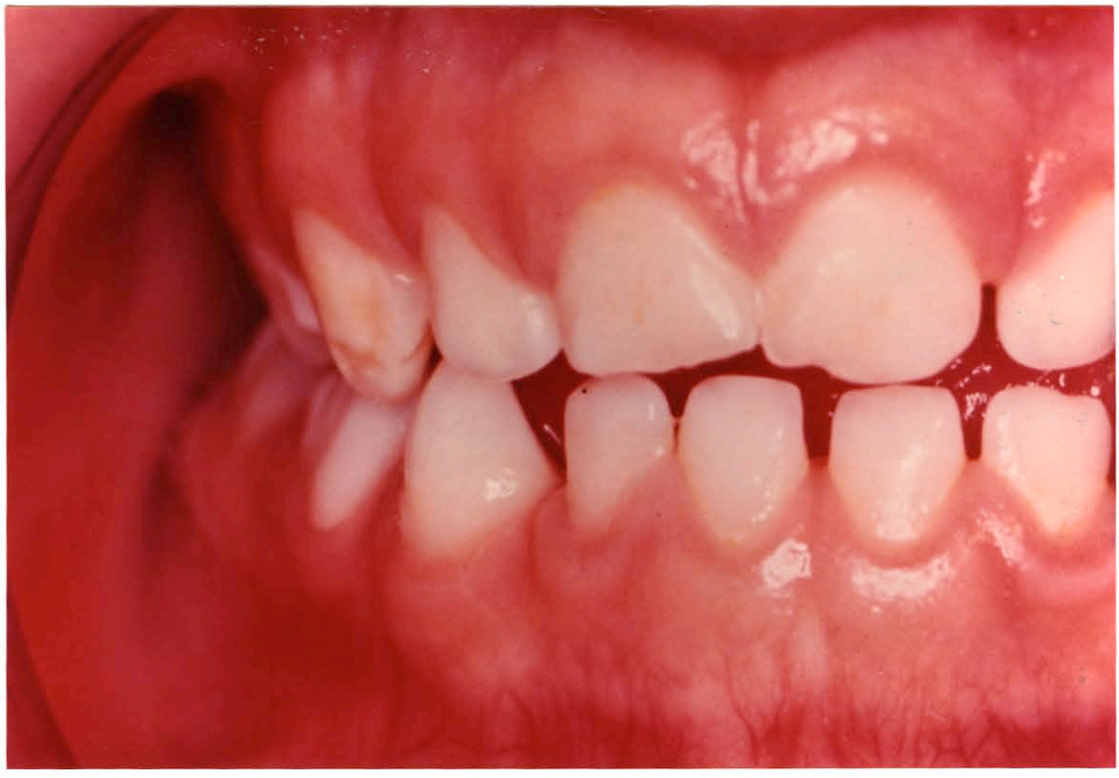


Illustration 3.3 Enamel hypoplasia with opacity occurring concurrently (HO) obvious on tooth 53

In the control group (Fig.3.5.2), enamel hypoplasia was found to have affected control children in very small numbers, 2 children (10%) in Exam III and only one child (4%) in Exam I. No children had hypoplasia in Exam II.

Enamel opacities were found in 5 children (45.5%) in Exam II with almost equal numbers of children affected by opacities in Exam I and III, that is 7 children (28.0%) and 6 children (30.0%) respectively.

HO affected the same two control children at all three examinations. Due to the different total numbers of control examined at Exam I, II and III the percentages were 8.0%, 18.2% and 10.0% respectively.

Table 3.5.2 provides comparisons of various types of enamel defects between the numbers of preterm and control children at each examination. The differences between the numbers of affected preterm and control children for each of the types of enamel defects are as follows. For enamel hypoplasia, the differences were tested to be statistically extremely significant in Exam I and III, with Exam II being statistically very significant.

However, for enamel opacities, the differences were not significant in Exam I and II but were extremely significant in Exam III (P=0.0001).

Table 3.5.2 Comparison of various types of enamel defects in the preterm and control children.

	Exam I		Exam II		Exam III	
	Preterm	Control	Preterm	Control	Preterm	Control
Total no. of children	25	25	19	11	25	20
No. of children (%) affected with						
Enamel Hypoplasia	17(68.0%)	1(4.0%)	10(52.6%)	0	16(64.0%)	2(10.0%)
(P-value)	<0.00001		0.001		0.00013	
Enamel Opacities	12(48.0%)	7(28.0%)	14(73.7%)	5(45.5%)	21(84.0%)	6(30.0%)
(P-value)	[N.S.]		[N.S.]		0.0001	
Hypoplasia with Opacity	0	2(8.0%)	3(15.8%)	2(18.2%)	12(48.0%)	2(10.0%)
(P-value)	[N.S.]		[N.S.]		0.0046	

(P-value) from Chi-square, χ^2 test
[N.S.] denotes P-value is statistically not significant

Similarly, for HO, the differences between the preterm and control were only statistically very significant in Exam III with Exam I and II having no statistically significant differences between the affected preterm and control.

3.5.2 The number of teeth affected by enamel defects in the preterms and controls.

Figs.3.5.3 shows the number of teeth affected by the various types of enamel defects, in the preterms and full term controls respectively.

The number of teeth of the preterm children (Fig.3.5.3) affected by enamel hypoplasia was almost similar in Exam I, II and III, that is , 43(11.4%), 36(9.2%) and 51(10.2%) respectively. In contrast, enamel opacities (Fig.3.5.3) affected 111(38.0%) teeth in Exam III and 48(12.2%) in Exam II and only 24(6.4%) in Exam I. Enamel opacities and hypoplasia occurring concurrently (HO), affected 29 (9.9%) in Exam III and 6 (1.5%) in Exam II. No teeth were affected by HO in Exam I.

The number of teeth of the control children (Fig. 3.5.4) affected by enamel hypoplasia, opacities, and HO were generally in very small numbers. Enamel hypoplasia was found in only 5 (1.3%) teeth at Exam III and 4 (0.8%) teeth at Exam I. No teeth were observed to be affected by hypoplasia in Exam II.Enamel opacities affected 12 (3.0%) in Exam III, 9 (1.9%) in Exam I and the same 9 (4.1%) teeth in Exam II. The percentages were different for Exam I and III due to the different total number of teeth examined as a group. HO affected the same 3 teeth at all

Fig.3.5.3 Percentage of Teeth in Preterm Children with Enamel Defects

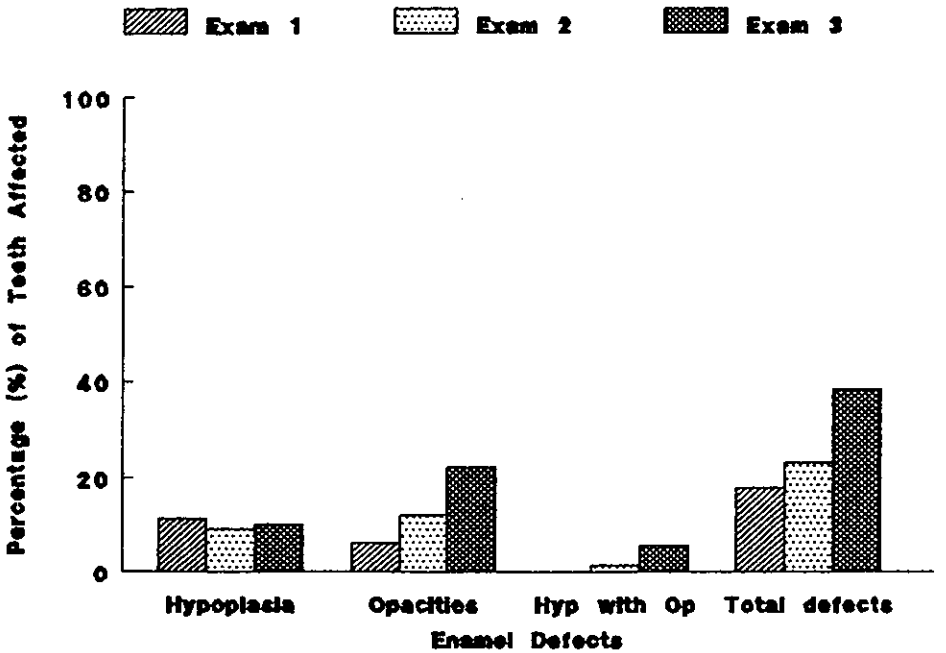
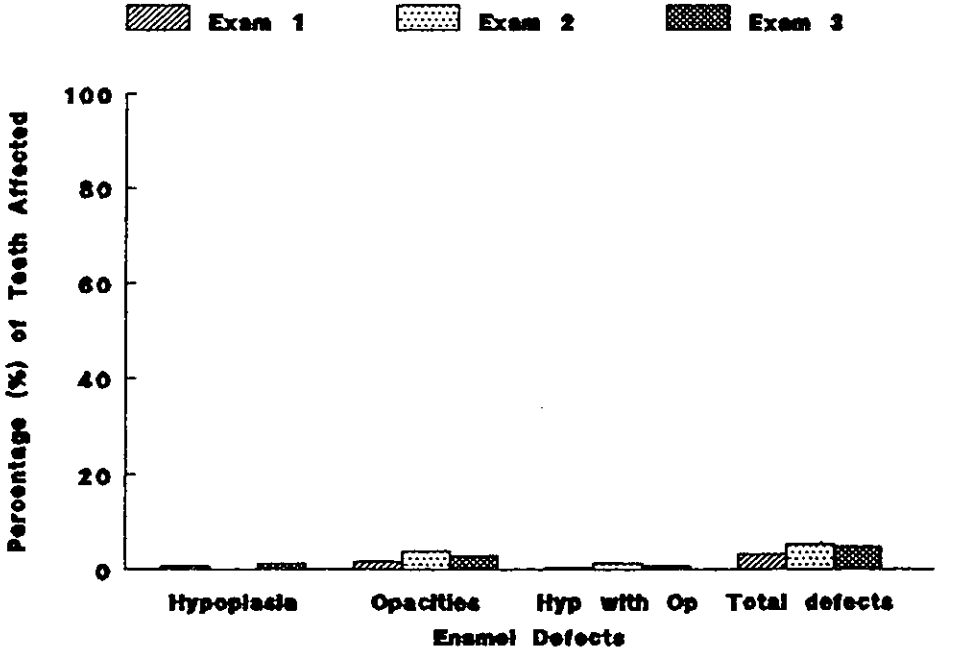


Fig.3.5.4 Percentage of Teeth in Control Children with Enamel Defects



three exams although the percentages were different at Exam I, II and III at 6.0%, 1.4% and 0.8% respectively, due to the different totals of teeth examined in the three examinations.

Table 3.5.3 provides comparisons of various enamel defects between the number of teeth affected in both preterm and control children at each examination. The differences between the percentages of affected preterm and control children for each of the types of enamel defects are as follows. For enamel hypoplasia, the differences were tested to be statistically extremely significant at Exam I, II and III.

Similarly, the differences in the number of affected teeth for enamel opacity were statistically very significant at Exam I and II with Exam III being extremely significant.

However, for hypoplasia with opacity occurring concurrently (HO) type of enamel defect, the differences in the number of affected teeth between preterm and control children were not significant at Exam I and II, but very significant in Exam III.

Table 3.5.3 Comparison of various types of enamel defects according to the number of teeth affected.

	Exam I		Exam II		Exam III	
	Preterm	Control	Preterm	Control	Preterm	Control
Total no. of teeth	377	476	392	220	499	399
No. of teeth (%) affected with						
Enamel Hypoplasia	43(11.4%)	4(0.8%)	36(9.2%)	0	51(10.2%)	5(1.3%)
(P-value)	<0.00001		<0.00001		<0.00001	
Enamel Opacity	24(6.4%)	9(1.9%)	48(12.2%)	9(4.1%)	111(22.2%)	12(3.0%)
(P-value)	0.001		0.001		<0.00001	
Hypoplasia with Opacity	0	3(0.6%)	6(1.5%)	3(1.4%)	29(5.8%)	3(0.8%)
(P-value)	0.38 [N.S.]		0.85 [N.S.]		0.0001	

(P-value) from Chi-square, χ^2 test
[N.S.] denotes P-value is statistically not significant

3.6 Prevalence of dental caries

Table 3.6.1 summarises the number of children and teeth affected by the decay and/or fillings in both the preterms and controls together with their respective mean d.m.f.t. scores.

Overall, dental caries prevalences were detected in almost similar percentages of children in both preterm and control groups. As tabulated, in Exam I, none of the total number of 25 preterm children presenting for examination, had caries, and only one of the 25 (4%) control children had caries. In Exam II, 4(21%) out of 19 preterm children had caries experience compared to 1 (9%) out of 11 control children. In Exam III, 7(28%) out of 25 of the preterm children had caries experience in comparison to 4(20%) out of 20 of the control children.
The differences between the numbers of caries affected preterm and control children however, were not statistically significant, at all examinations (P-values at Exam I, II, and III were 1.0, 0.6 and 0.8 respectively).

Looking at the numbers of affected teeth of both groups, the picture is fairly similar to that of the numbers of affected children.
At Exam I, caries did not affect any of the teeth in the preterm group and likewise in the control group, only 1(0.2%) out of 476 teeth was affected with caries.
At Exam II, 11(2.8%) of the 392 teeth in the preterms were carious and/or filled whereas 5(2.3%) of the 220 teeth in the controls were carious.

Table 3.6.1 Dental caries in the preterm and control children

	Exam I		Exam II		Exam III	
	Preterm	Control	Preterm	Control	Preterm	Control
Mean Age ±SD(months)	27.4 ±4.5	32.0 ±3.1	41.3 ±6.3	45.4 ±3.8	51.9 ±5.1	52.5 ±4.2
Total no. of children	25	25	19	11	25	20
Total no. of teeth	377	476	392	220	499	399
Decayed &/or Filled (d.f.)						
Affected children(%)	0	1 (4%)	4(21%)	1(9%)	7(28%)	4(20%)
(P-value)·	1.0 [N.S.]		0.6 [N.S.]		0.8 [N.S.]	
Affected teeth(%)	0	1(0.2%)	11(2.8%)	5(2.3%)	20(4.0%)	28(7.0%)
(P-value)·	1.0 [N.S.]		0.8 [N.S.]		0.1 [N.S.]	
Mean d.m.f.t.±SD	0	0.0± 0.2	0.6±1.4	0.5±1.5	0.8±1.5	1.4±3.2
(P-value)··	[N.A.]		0.9 [N.S.]		0.8 [N.S.]	
Range	0	0-1	0-5	0-5	0-5	0-12
No.(%) of caries-free children	25(100%)	24(96.0%)	15(79.0%)	10(91.0%)	18(72.0%)	16(80.0%)
(P-value)·	N.A.		0.157 [N.S.]		0.730 [N.S]	

(P-value)· from Fisher's Exact Test
(P-value)·· from *t*-test (unpaired)
[N.A.] denotes not applicable
[N.S.] denotes P-value is statistically not significant

At the final examination (Exam III) at the mean ages of 51.9 and 52.5 months for the preterms and controls respectively, 20(4.0%) of the 499 teeth in the preterms were decayed and/or filled and 28(7.0%) in the controls were decayed and/or filled.

The differences between the numbers of decayed and/or filled teeth of affected preterm and control children were again not statistically significant, at all examinations, that is P-values at Exam I, II, and III were 1.0, 0.8 and 0.1 respectively.

The mean d.m.f.t. ± SD score at Exam I were 0 for both preterms and controls. At Exam II the mean scores were 0.6 ± 1.4 for the preterms and 0.5 ± 1.5 for the controls. Similarly, at Exam III, the mean scores were 0.8 ± 1.5 for the preterms and 1.4 ± 3.2 for the controls. The differences in the mean d.m.f.t. scores of the preterm and control children were again not statistically significant, at all examinations, with P-values at Exam II and III being 0.9 and 0.8 respectively. At Exam I the 0 mean scores results in no P-value.

The ranges for the d.m.f.t. scores at Exam I were 0 and 0 - 1 for the preterm and control children respectively. At Exam II, both preterm and control children showed a similar range of d.m.f.t. scores (0 - 5). However, at Exam III, the control children had a broader range of d.m.f.t. scores (0 - 12) compared to the preterm children of 0 - 5.

Looking at the data from the caries-free perspective, the numbers of

caries-free children at Exam I were 25(100%) and 24(96%) for the preterm and control children respectively. At Exam II, the caries-free numbers were 15(79%) and 10(91%) for the preterm and control children respectively. Lastly, at Exam III, the caries-free numbers were 18(72%) and 16(80%) for the preterm and control children respectively. The differences comparing the preterm and control groups at each examination however, were not significant.

3.6.1 The number of preterm and control children affected by caries

The number of preterm and full-term control children affected by caries is illustrated in Figs. 3.6.1 and 3.6.2 respectively. These graphs also include control children with decayed(d) or filled(f) components besides the total number of children affected with decay and/or fillings.

In the preterm group (Fig.3.6.1), the decayed (d) teeth were detected in 4 (21.1%) of the 19 children at Exam II and 4 (16.0%) of the 25 children at Exam III. The 4 decay-affected children seen at Exam II and III were the same but yield different percentages because of the different total numbers of children examined. No children with decay were detected in Exam I. The filled (f) component was observed only at the final exam (Exam III) in 4 (16.0%) of the 25 preterm children.

The total number of preterm children affected by decay &/or fillings were 7(28%) instead of 8 because one preterm child had both decay and fillings meaning that this child was included in the d-affected and also f-affected preterm groups. In short, the d and f-affected groups were not mutually exclusive.

Fig.3.6.1 Percentage of preterm children with Dental Caries

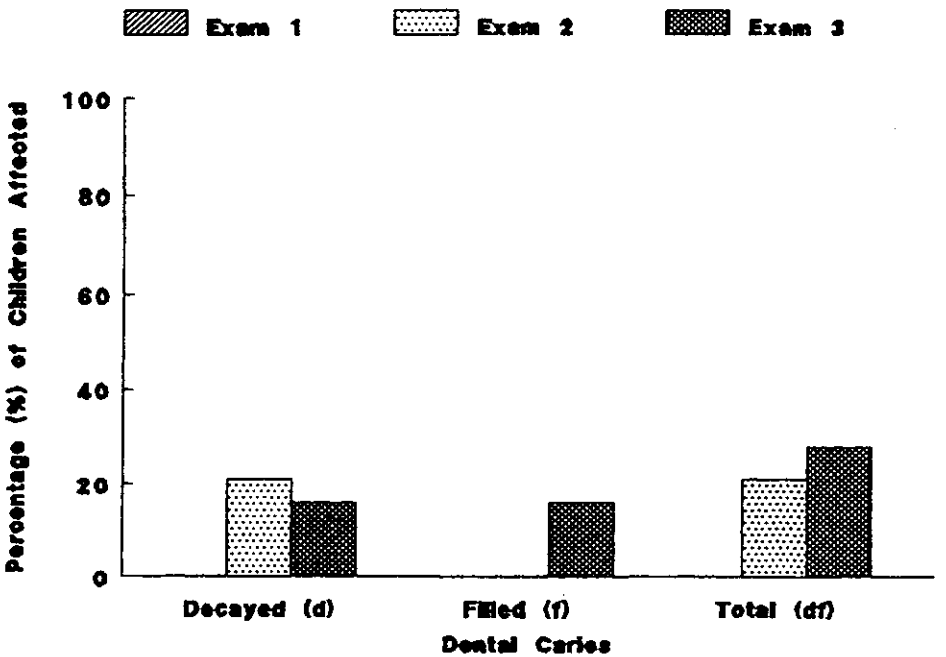
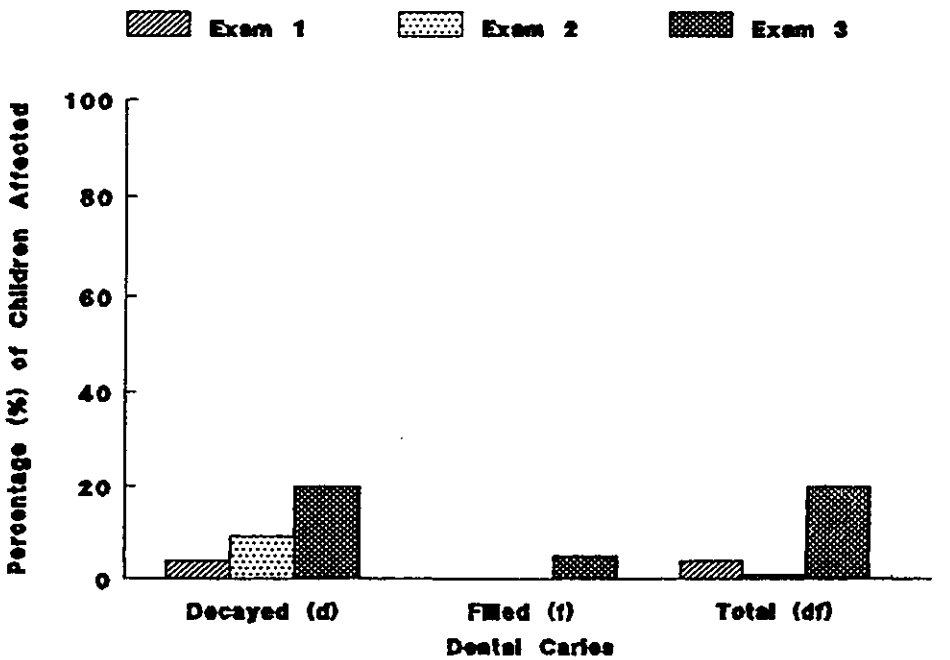


Fig.3.6.2 Percentage of control children with Dental Caries



In the control group (Fig. 3.6.2), the decayed (d) component was found in one (4%) of the 25 children in Exam I, one (9.1%) of the 11 children in Exam II and 4 (20%) of the 20 children in Exam III.
The f-component was only observed in Exam III with one (5%) of the 20 children affected.

The total number of affected control children were 4(20%) instead of 5 due to the similar situation as of the preterm where one child had both d and f component and was counted once in the d-affected group and again in the f-affected group.

Table 3.6.2 provides comparisons of d and f-components between the percentages of affected preterm and control children at each examination. The differences between the percentages of affected preterm and control children for each of the d and f-component types were all not statistically significant.

Table 3.6.2 Comparisons of d and f-components between the percentages of affected preterm and control children.

	Exam I		Exam II		Exam III	
	Preterm	Control	Preterm	Control	Preterm	Control
Total no. of children	25	25	19	11	25	20
No. of affected children (%)						
Decayed (d)	0	1(4.0%)	4(21.1%)	1(9.1%)	4(16.0%)	4(20.0%)
(P-value)*	1.000[N.S.]		0.735[N.S.]		0.970[N.S.]	
Filled (f)	0	0	0	0	4(16.0%)	1(5.0%)
(P-value)*	[N.A.]		[N.A.]		0.490[N.S.]	

(P-value)* from Fisher's Exact Test
[N.A.] denotes not applicable
[N.S.] denotes P-value is statistically not significant

3.6.2 The number of teeth affected by caries in the preterm and control children.

Figs.3.6.3 and 3.6.4 illustrate the number of teeth with decayed (d) and filled (f) components besides the total number of affected teeth in the preterm children and control children respectively.

In the preterm group (Fig.3.6.3), the decayed (d) teeth were detected in 11 (2.8%) of the 392 teeth at Exam II, and 10 (2.0%) of the 499 teeth at Exam III. No teeth had decay at Exam I.
The filled (f) component was observed in the final Exam III in 10 (2.0%) of the 499 teeth.

In the control group (Fig. 3.6.4), decay was found in 1(0.2%) of the 476 teeth at Exam I, 5(2.3%) of 220 teeth at Exam II and 23(5.8%) of the 399 teeth at Exam III. Filled teeth were observed at Exam III in 5(1.3%) of the 399 teeth in the control children. No teeth were filled in Exam I and II.

Fig.3.6.3 Percentage of Teeth in Preterm Children with Dental Caries

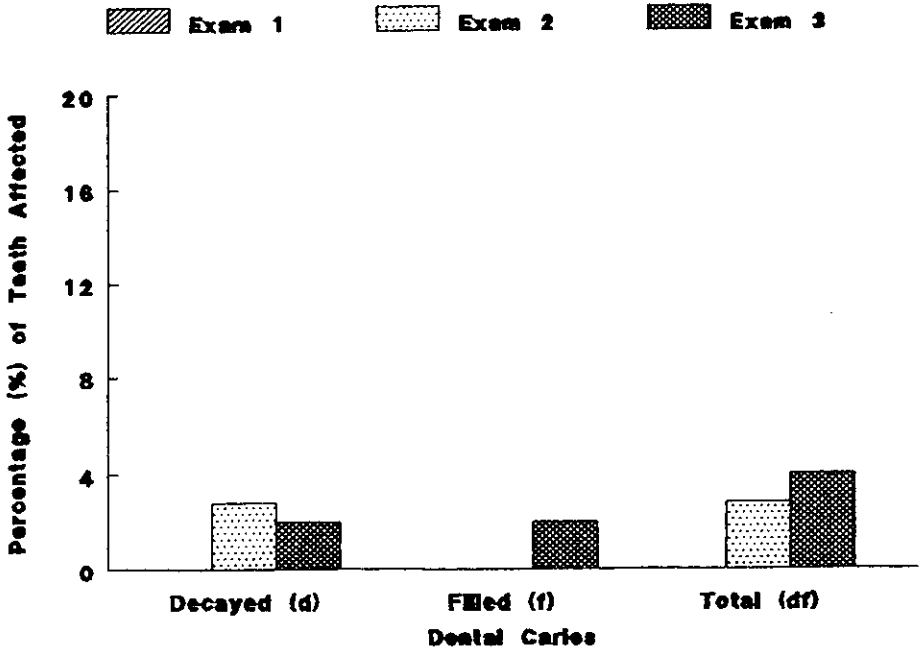


Fig.3.6.4 Percentage of Teeth in Control Children with Dental Caries

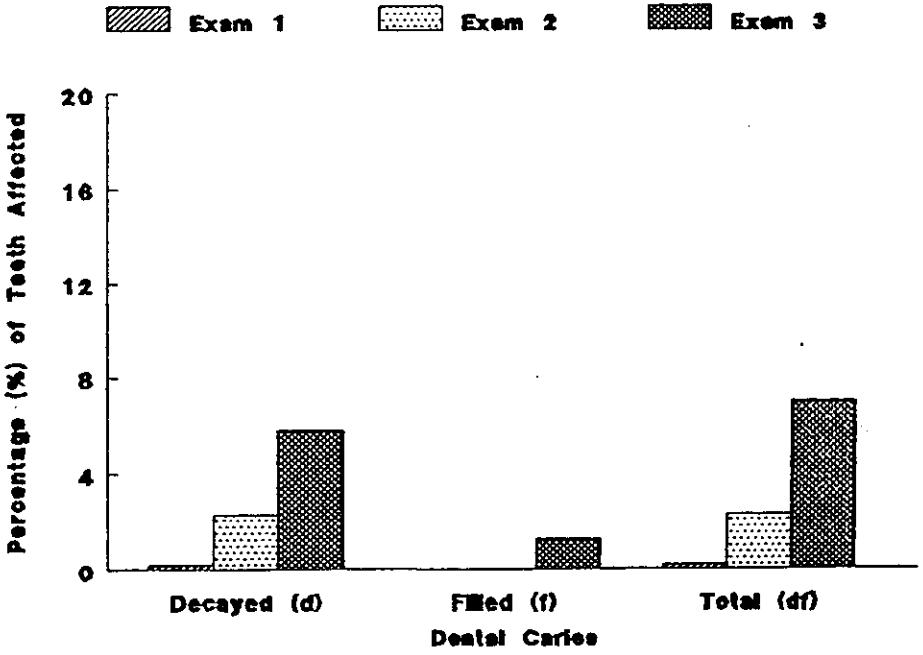


Table 3.6.3 provides comparisons of d and f-components between the numbers of teeth affected in both the preterm and control children at each examination. The differences between the numbers of teeth affected in the preterm and control children for each of the d and f-component types are as follows. For decayed teeth, the differences between the percentages in preterm and control children were tested to be statistically not significant for Exam I and II, but were very significant for Exam III.

Table 3.6.3 Comparisons of d and f-components between the numbers and percentages of affected teeth in the preterm and control children.

	Exam I		Exam II		Exam III	
	Preterm	Control	Preterm	Control	Preterm	Control
Total no. of teeth	377	476	392	220	499	399
No. of teeth (%) affected with						
Decayed (d)	0	1(0.2%)	11(2.8%)	5(2.3%)	10(2.0%)	23(5.8%)
(P-value)·	0.90 [N.S.]		0.90 [N.S.]		0.005	
Filled (f)	0	0	0	0	10(2.0%)	5(1.3%)
(P-value)·	[N.A.]		[N.A.]			0.54 [N.S.]

(P-value)· from Fisher's Exact Test
[N.A.] denotes not applicable
[N.S.] denotes P-value is statistically not significant

3.7 Distribution of teeth affected by enamel defects and dental caries according to tooth type

The distribution of teeth affected by enamel defects and dental caries according to their tooth type is shown in Tables 3.7.1 & 3.7.2 for the preterm and control children respectively.

In the preterm children the highest percentage of enamel defects was noted in the primary second molars, 52% (both mandibular and maxillary); followed by the primary maxillary incisors (49.5%), the primary first molars (44% of mandibular and 42% of maxillary) and primary canines (32% of mandibular and 30% of maxillary). The least affected teeth were the primary mandibular incisors (13%).

Dental caries in the preterm children was most frequent again in the primary second molars (18% of the maxillary and 10% of the mandibular), followed by the primary maxillary first molars (4%), with the primary mandibular first molars, maxillary canines and incisors all at the same frequency of 2%. The primary mandibular canines and incisors were not affected by caries.

In the controls (Table 3.7.2), the highest percentage of enamel defects was noted in the primary canines (15% of maxillary and 10% of mandibular); followed by the primary maxillary incisors (8.9%), the primary second molars (5% of mandibular and 2.5% of maxillary). The primary first molars and mandibular incisors were not affected.

Dental caries in the control children was most frequent in the primary first molars, 15% (both the maxillary and mandibular), followed by the primary maxillary second molars and mandibular canines at equal frequency

Table 3.7.1
Enamel Defects and Caries in the preterm children by tooth type

	Enamel Defects		Caries developing at subsequent exam		
	Present	Absent	Present	Absent	P-value
	Number (%)		Number (%)		
Primary Second Molars					
maxillary, n = 50	26 (52)	24 (48)	9 (18)	41 (82)	P < 0.01
mandibular, n = 50	26 (52)	24 (48)	5 (10)	45 (90)	N.S.
Primary First Molars					
maxillary, n = 50	21 (42)	29 (58)	2 (4)	48 (96)	N.S.
mandibular, n = 50	22 (44)	28 (56)	1 (2)	49 (98)	N.S.
Primary Canines					
maxillary, n = 50	15 (30)	35 (70)	1 (2)	49 (98)	N.S.
mandibular, n = 50	16 (32)	34 (64)	0	50 (100)	-
Primary Incisors					
maxillary, n = 99	49 (49.5)	50 (50.5)	2 (2)	97 (98)	N.S.
mandibular, n = 100	13 (13)	87 (87)	0 (0)	100 (100)	-
TOTAL n = 499	188 (37.7)	311 (62.3)	20 (4)	479 (96.0)	

Overall χ^2 test is not applicable

Table 3.7.2
Enamel Defects and Caries in the control children by tooth type

	Enamel Defects		Caries developing at subsequent exam	
	Present Number (%)	Absent	Present Number (%)	Absent
Primary Second Molars				
maxillary, n = 40	1 (2.5)	39 (97.5)	4 (10)	36 (90)
mandibular, n = 40	2 (5)	38 (95)	2 (5)	38 (95)
Primary First Molars				
maxillary, n = 40	0 (0)	40 (100)	6 (15)	34 (85)
mandibular, n = 40	0 (0)	40 (100)	6 (15)	34 (85)
Primary Canines				
maxillary, n = 40	6 (15)	34 (85)	0 (0)	40 (100)
mandibular, n = 40	4 (10)	36 (90)	4 (10)	36 (90)
Primary Incisors				
maxillary, n = 79	7 (8.9)	72 (91.1)	6 (7.6)	73 (92.4)
mandibular, n = 80	0 (0)	80 (100)	0 (0)	80 (100)
TOTAL n = 399	20 (5)	379 (95)	28 (7.0)	371 (93)

Overall χ^2 test is not applicable

of 10%, with the primary maxillary incisors at 7.6% and the primary mandibular second molars at 5%. The primary maxillary canines and mandibular incisors were not affected by caries.

3.8 Association of Enamel Defects and Dental Caries

3.8.1 In the Preterm children

Following on from the data obtained for enamel defects and caries prevalence, in order to assess if a significant association existed between enamel defects and caries, enamel defects were monitored longitudinally. Enamel defects presenting with and without dental caries were compared. Table 3.8.1 shows the association of enamel defects and caries at examinations I, II and III, in the preterms. The results indicate that caries tended to occur more on teeth with enamel defects (Illustration 3.8) than on teeth without enamel defects. The relationship between enamel defects and caries was statistically extremely significant at Exam II and III ($P<0.00001$ and $P=0.0001$ respectively). At Exam I these children were not observed to experience caries at a mean age of 27.4 ±4.5 months.

Our longitudinal observation was that if caries was to occur on these hypoplastic teeth, it occurred very shortly after eruption, thus making it difficult to catch these teeth at a stage totally unaffected by decay at the scheduled examinations. When seen these teeth appeared to show both caries and enamel defects. There were 11 such affected teeth out of the 16 carious teeth in the preterm children at Exam III and 9 out of 10 in Exam II. These caries affected teeth were mainly the 2nd primary molars as mentioned in Chapter 3.7 p.75. Nonetheless the author observed

Table 3.8.1 Association of Dental Caries with Enamel Defects by the number of teeth in the preterm group

		Dental Caries Number (%)			
		Present	Absent	Total	P-value
Enamel Defects in Exam I	Present	0 (0.0)	67 (17.8)	67 (17.8)	N.A.
	Absent	0 (0.0)	310 (82.2)	310 (82.2)	
	Total	0 (0.0)	377 (100)	377 (100)	
Enamel Defects in Exam II	Present	10 (2.6)	80 (20.4)	90 (23.0)	P < 0.00001
	Absent	1 (0.3)	301 (76.8)	302 (77.0)	
	Total	11 (2.9)	381 (97.2)	392 (100)	
Enamel Defects in Exam III	Present	16 (3.2)	172 (34.5)	188 (37.7)	P = 0.00010
	Absent	4 (0.8)	307 (61.5)	311 (62.3)	
	Total	20 (4.0)	479 (96.0)	499 (100)	

The above P-values (two tailed) were calculated using the Fisher's Exact Test

N.A. denotes statistical test was not applicable

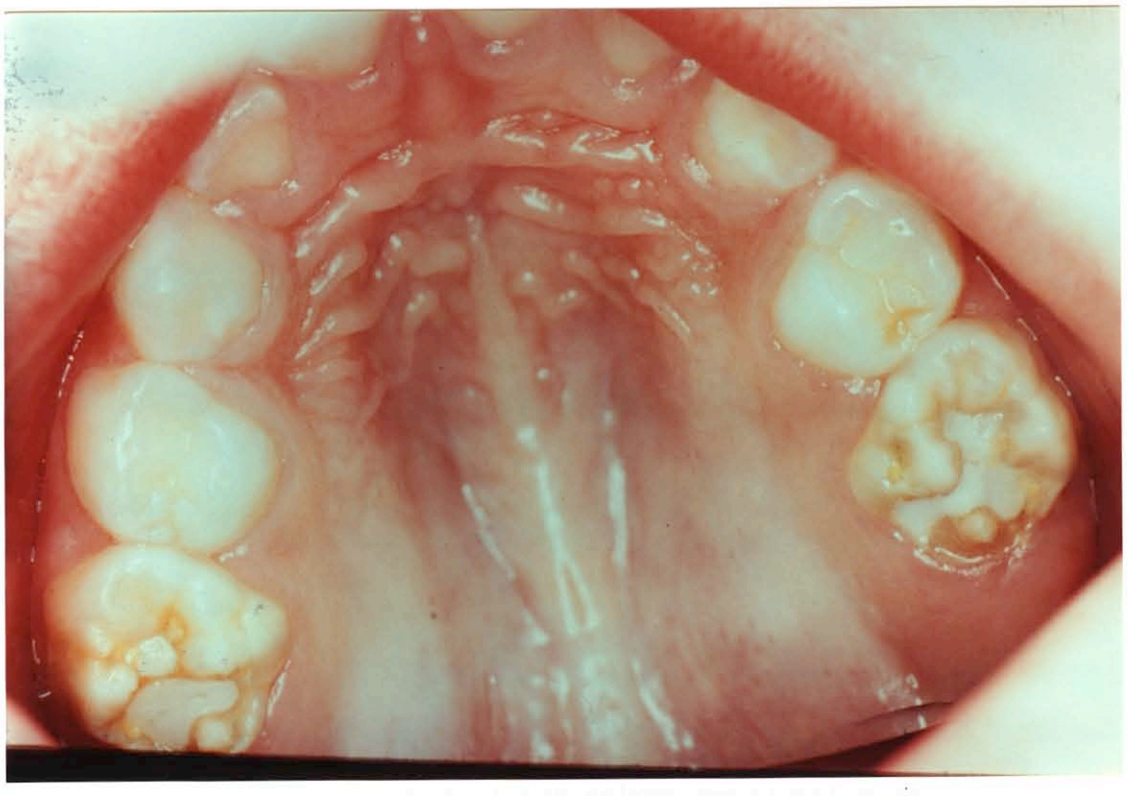


Illustration 3.8 Glass ionomer interim restorations on carious teeth 55 and 65 affected by enamel defects

enamel defects being present on the same teeth and/or surfaces where caries occurred.

Also found were a total of 6 teeth in 3 patients which were not diagnosed to be affected by enamel defects but subsequently showed enamel defects and caries. There were two upper central incisors in one patient not diagnosed with enamel defects (opacities) in Exam I, but subsequently developed two interproximal caries and buccal opacities in Exam II.

Similarly, there was a tooth in a patient not diagnosed to be with opacities in Exam I, but subsequently showed opacities and caries in Exam III with patient not attending Exam II. Yet another patient had three teeth (2nd molars) not diagnosed to be affected by enamel defects at Exam II and were not erupted in Exam I but subsequently had occlusal caries at Exam III with marked opacities covering two-thirds of the occlusal surfaces.

Of interest was the one tooth (upper left primary canine) with buccal opacity in the middle-third surface of a patient in Exam I which subsequently became carious in this area in Exam II and was filled by Exam III. This area is a highly unlikely place for caries to occur in any situation. The child concerned had three other teeth (2nd molars) not previously diagnosed to be affected by enamel defects at Exam II, with these teeth unerupted in Exam I and subsequently experiencing occlusal caries and marked opacities on the surfaces of these teeth.

3.8.2 In the control children

The association of enamel defects and caries in the control children is

Table 3.8.2 Association of Dental Caries with Enamel Defects by the number of teeth in the full-term, control group

		Dental Caries (Percentages, %)			
		Present	Absent	Total	P-value
Enamel Defects in Exam I	Present	0 (0.0)	16 (3.4)	16 (3.4)	P = 1.0000
	Absent	1 (0.2)	459 (96.4)	460 (96.6)	
	Total	1 (0.2)	475 (99.8)	476 (100.0)	
Enamel Defects in Exam II	Present	0 (0)	12 (5.5)	12 (5.5)	P = 0.9999
	Absent	5 (2.3)	203 (92.3)	208 (94.6)	
	Total	5 (2.3)	215 (97.8)	220 (100.1)	
Enamel Defects in Exam III	Present	1 (0.3)	19 (4.8)	20 (5.1)	P = 1.0000
	Absent	27 (6.8)	352 (88.2)	379 (95.0)	
	Total	28 (7.1)	371 (93.0)	399 (100.1)	

The above P-values (two tailed) were calculated using the Fisher's Exact Test

shown not to be statistically significant (Table 3.8.2 p.82). The reason for this is because of the small number of teeth affected with enamel defects. For example, only twenty-eight (7.1%) out of the total of 399 teeth were affected in Exam III.

3.9 Dental caries and the different types of enamel defects

In order to assess as to whether a particular type of enamel defect was more susceptible than other types of enamel defects to dental caries, teeth affected with different types of enamel defects were compared in relation to caries being present or absent. Table 3.9.1 shows the relationship of various types of enamel defects to caries in the preterm children and Table 3.9.2 shows that for the controls.

In the preterm children (Table 3.9.1), statistical testing of the data showed that the only type of enamel defect being associated with caries in the preterm children was the opacities type. The P-value for the opacities alone group was less than 0.00001 indicating extreme significance. The hypoplasia and HO types had non-significant P-values of 0.09 and 0.62 respectively.

In the control children (Table 3.9.2), no association was found between the various types of enamel defects and caries. The non-significant P-values for the hypoplasia, opacities and HO types of enamel defects were 0.31, 0.62 and 1.00 respectively.

Table 3.9.1 The association of various types of enamel defects to caries in the preterm children by the number of teeth.

Enamel Defects(%)	Caries(%) Present	Absent	Total	P-value (From Fisher's Exact Test)
None	4 (0.8)	304 (60.9)	308(61.7)	
Hypoplasia alone	0 (0)	51 (10.2)	51(10.2)	P = 0.09 N.S.
Opacities alone	14 (2.8)	97 (19.4)	111(22.2)	P <0.00001
Hypoplasia with Opacity occurring concurrently (HO)	2 (0.4)	27 (5.4)	29(5.8)	P = 0.62 N.S.
Total	20(4.0)	479(95.9)	499(99.9)	

Overall χ^2 test is not applicable

Table 3.9.2 The association of various types of enamel defects to caries in the control children by the number of teeth.

Enamel Defects(%)	Caries(%) Present	Absent	Total	P-value (From Fisher's Exact Test)
None	27 (6.8)	352 (88.2)	379 (95.0)	
Hypoplasia	1 (0.3)	4 (1.0)	5 (1.3)	P = 0.31 N.S.
Opacities	0 (0)	12 (3.0)	12 (3.0)	P = 0.62 N.S.
Hypoplasia with Opacity occurring concurrently (HO)	0 (0)	3 (0.8)	3 (0.8)	P = 1.00 N.S.
Total	28(7.1)	371(93.0)	399(100.1)	

Overall χ^2 test is not applicable

3.10 Other factors influencing dental caries

In investigating caries in the preterm children, various influencing factors on caries were looked at. These included the *Strep. mutans* level, plaque level, brushing frequency, fluoride exposure, sugar exposure and socioeconomic status (Table 3.10.1).

3.10.1 *Strep. mutans* score

The microbiological indicator for the level of *Strep. mutans* infection, the *Strep. mutans* score was used on each patient at Exam III. In the preterms, the *Strep. mutans* scores were variable. Three out of 7 caries-present preterms had a score of 2 and the others had a score of 0. Of the 19 caries-free preterms, 13 had a score of 0 with the others being distributed over the scores of 1-3. On the other hand, most of the caries-free controls (10 out of 11) had a score of 0 in contrast to all caries-present controls having a score of 2 or 3.

3.10.2 Plaque score

Five out of the 8 caries-present preterms and 10 out of 17 caries-free preterms had plaque score of I. In the controls, 13 of the 20 caries-free children had a score of I whereas 3 out of the 4 caries-present controls had a score of II with the remaining one child having a score of III.

Table 3.10.1 Factors Influencing Dental Caries

	Number of Children (%)			
	Preterms		Full-term Controls	
	Caries Present	Caries Free	Caries Present	Caries Free
Strep. mutans score				
0	4(15.4)	13(50.0)	0(0)	10(71.4)
1	0(0)	3(11.5)	0(0)	1(7.1)
2	3(11.5)	2(7.7)	3(21.4)	0(0)
3	0(0)	1(3.8)	1(7.1)	0(0)
P-value	N.A.		N.A	
P-value (Preterm vs Control caries gps)	N.A.			
Plaque score				
0 - 25%:I	5(20)	10(40)	0(0)	13(54.2)
more than 25 - 50%:II	2(8)	6(24)	3(12.5)	6(25.0)
more than 50 - 75%:III	1(4)	1(4)	1(4.2)	0(0)
more than 75% :IV	0(0)	0(0)	0(0)	1(4.1)
P-value	N.A.		N.A.	
P-value (Preterm vs Control caries gps)	N.A.			
Brushing History (mean frequency/day)				
less than 1	0(0)	2(7.7)	0(0)	0(0)
1 to less than 2	1(3.8)	10(38.5)	4(14.8)	11(40.7)
2 to less than 3	6(23.1)	7(26.9)	0(0)	11(40.7)
3 and more	0(0)	0(0)	1(3.7)	0(0)
P-value	N.A.		N.A.	
P-value (Preterm vs Control caries gps)	N.A			
Fluoride Exposure				
Yes	5(19.2)	10(38.5)	1(3.7)	6(22.2)
No	2(7.7)	9(34.6)	4(14.8)	16(59.3)
P-value	N.S(p=0.580)		N.S(p=0.818)	
P-value (Preterm vs Control caries gps)	N.A.			
Sugar Intake (mean daily frequency)				
1 to less than 2	0(0)	5(23.8%)	0(0)	2(22.2%)
2 to less than 3	3(14.3%)	3(14.3%)	0(0)	3(33.3%)
3 to less than 4	3(14.3%)	5(23.8%)	1(11.1%)	2(22.2%)
4 to less than 5	0(0)	2(9.5%)	0(0)	1(11.1%)
P-value	N.A			N.A.
P-value (Preterm vs Control caries gps)	N.A.			

3.10.3 Toothbrushing history

The mean brushing frequency per day in the caries-present preterm children was mainly twice a day (6 out of the 7 children) with the caries-free children being either once (10 of the 19 children) or twice (7 out of 19 children) a day. Most caries-present controls (4 of 5 children) had a mean brushing frequency of once a day with caries-free controls brushing either once (11 of 22 children) or twice (11 of 22 children) a day.

3.10.4 Fluoride Exposure

Interestingly, in the preterms, 5 of the 7 children with caries had history of fluoride supplementation. The P-value between the caries-present and absent preterms was not significant (P=0.580). In the caries-present controls, fluoride exposure was only limited to 1 out of 5 children. Again the P-value between the caries-present and caries-free controls was not significant (P=0.818).

3.10.5 Sugar exposure

The mean daily frequency based on the diet charts collected at each examination for the caries-present preterms was in the 2 to less than 3 times per day range, for 3 out of 6 children and 3 to less than 4 times per day range, for the other 3 of 6 children. Caries-free children had frequency ranging from 1 to less than 5.

In the controls the caries-present children had a mean of 3 to less than 4 times per day whereas the caries-free controls were distributed almost evenly amongst all the 4 classes.

3.10.6 Socioeconomic status (S.E.S.)

The socioeconomic status of all participating children are shown in Table 3.10.2. In the preterm, 4 of the 7 caries-present children were in S.E.S. I, and with one child in S.E.S. III and remaining 2 children in S.E.S. IV. Eight caries-free children were in S.E.S. I, 3 in S.E.S. II and 7 caries-free children in S.E.S. III. There was no association of caries-present preterms with any particular S.E.S. group.

In the controls, 1 of the 5 caries-present children was in S.E.S. I, 3 in S.E.S. III and one child in S.E.S. IV. Eight of the 22 caries-free children were in S.E.S. I, 2 in S.E.S. II, 7 in S.E.S. III and 5 in S.E.S. IV.

Comparing caries-present preterms and controls according to their social classes, it can be seen that more than half (4 of the 7) caries-present preterms were from S.E.S. I, the highest S.E.S. group compared to only 1 out of the 5 caries-present controls.

Table 3.10.2 Socioeconomic Status (S.E.S.) and Dental Caries in the preterm and control children

	Number of Children (%)			
	Pre term		Full term Control	
Total no. of children	n = 25		n = 25	
	Caries Present	Caries Free	Caries Present	Caries Free
S.E.S.				
I [HIGH]	4 (16)	8 (32)	1 (4)	8 (32)
II [MIDDLE]	0 (0)	3 (12)	0 (0)	2 (8)
III [LOW]	1 (4)	7 (28)	3 (12)	7 (28)
IV [OTHERS]	2 (8)	0 (0)	1 (4)	5 (20)
P - value	N.A.		N.A.	
P - value (Preterm vs control caries groups)	N.A.			

3.11 Occlusion

Table 3.11.1 provides data on the occlusion of the preterm and control children. The preterm children had a mean overjet of 3.1±2.2mm and control children had a mean of 2.7±1.4mm (P = 0.43). The mean overbite (as a percentage of the mandibular incisor length) being 39 ±36.4% and 38 ±42.2% for the preterm and control children respectively (P = 0.93).

The investigation on the occlusal relationship gave the following results: For the molar-relationship, 21(80.8%) of the 26 preterm children had a mesial step relationship, 4(15.4%) a distal step relationship and 1(3.8%) with a flush terminal plane. In the control children,15(55.6%) had a mesial step, 6(22.2%) a distal step, and 6(22.2%) a flush terminal plane. Although there were differences in the percentages obtained comparing the preterm and the control children the P-values were not significant. For the canine-relationship the percentages were fairly similar. A Class I canine-relationship was found in 21(80.8%) of the preterms, Class II in 4(15.4%) and Class III in 1(3.8%) of preterm children. In the controls, 17(63.0%) had a Class I canine-relationship, 9(33.3%) a Class II and 1(3.7%) a Class III. Comparing the percentages obtained from the preterm and control children yield non-significant P-values.

Unilateral lingual cross-bite was present in 4(15.4%) and 4(14.8%) of preterm and control children respectively (P = 1.00).
Interestingly, retroclined lower incisors were found in 7.7% (two children) and crowding of the lower incisors in 11.5% (three children) of the preterm group but no such conditions were noted in the controls. These five preterm children in all, had a dental Class I molar and canine-relationships.

Table 3.11.1 Occlusion of the preterm and control children studied

	VLBW n = 26	Controls n = 27	P-value
Mean overjet (±SD)mm	3.1 ± 2.2	2.7 ± 1.4	t- test(unpaired) P = 0.43 (N.S.)
Mean overbite (±SD) (% of mandibular incisor length)	39 ± 36.4	38 ± 42.2	t-test(unpaired) P = 0.93 (N.S.)
Molar relationship	n (%)	n (%)	χ^2-test
Mesial step	21 (80.8)	15 (55.6)	P = 0.09 (N.S.)
Distal step	4 (15.4)	6 (22.2)	P = 0.78 (N.S.)
Flush terminal plane	1 (3.8)	6 (22.2)	P = 0.12 (N.S.)
Canine relationship			Fisher's Exact test
Cl I	21 (80.8)	17 (63.0)	P = 0.22(N.S.)
Cl II	4 (15.4)	9 (33.3)	P = 0.20(N.S.)
Cl III	1 (3.8)	1 (3.7)	P = 1.00(N.S.)
Cross-bite			
Lingual (unilateral)	4 (15.4)	4 (14.8)	P = 1.00(N.S.)
Buccal	0	0	-
Other occlusal anomalies			Fisher's Exact test
Retroclined lower incisors	2* (7.7)	0	P = 0.24(N.S.)
Crowding of lower incisors	3* (11.5)	0	P = 0.11(N.S.)

* found in patients with dental Class I occlusion

Chapter 4 Discussion

4.1 Introduction

This study is the first reported longitudinal study on developmental enamel defects and caries in the preterms. The results are in agreement with the findings by several other researchers (refer list of Table 1.1 p.15), in that enamel defects are found in higher prevalence in the preterm children compared to control full-term children. The author found prevalence rates of 88.0%, 94.7% and 96.0% at Exam I, II & III, respectively in the VLBW preterm children.

More importantly, the findings described in this thesis do lend support to the hypothesis that enamel defects, in particular, the enamel defects of the opacity type predisposed the preterm children to higher caries risk.

Furthermore, this study is the first to describe a caries pattern in the preterm children not previously reported in the dental literature. This unique pattern involved caries being observed on the primary second molars most frequently, followed by the first maxillary molars and with the maxillary incisors and canines at equal third ranking (Table 3.7.1 p.76). The caries pattern was not related to the chronology of tooth eruption, or length of time the teeth had been present in the oral cavity.

This chapter discusses the findings of the study, in the light of current understanding of enamel defects and caries in the preterm children.

4.2 Subjects in Study

Preterm children:

In retrospect, judging from the results obtained, it is in the opinion of the author that the VLBW preterm sample was representative of the VLBW preterm children population. Many of the parents of these preterm children having regularly attended the Growth and Development Clinic at the Mater Hospital, with their preterm children were particularly aware of possible dental problems and were enthusiastic in participating in the study. The moderate participation rate of 59% (27 preterm children) is likely to be due to the fact that many of these preterm (6 of the 11 'non-respondents') were residing in places far away from Brisbane.

Control Children

Looking at the caries pattern, and S.E.S. and the low participation rate (28%) in hind sight, it is in the opinion of the author that the control population may <u>not</u> be representative of the population at large with respect to caries experience. The reason for this will be elaborated further in Chapter 4.6 p.106. Therefore, direct comparison of the caries experience, may not be valid as there is a bias towards a control with a higher caries experience.

The attendance rate in the preterms was satisfactory with 96% of all preterm attending the final examination. The attrition rate was thus very low. However while the controls' participation rate was less at 74% in the 3rd examination, the attrition rate was still considered acceptable for a longitudinal study.

Looking at the demography of the participants, the control children matched the preterm children closely for their sexes, races and date of birth. Children selected for this study were also comparable in racial composition. They comprised predominantly of Caucasian children (25 of 26 children) and one Chinese, for both the preterm and control groups. Interestingly, the spread of socioeconomic groups was also fairly similar.

As illustrated in Table 3.4.1 (p.50), the preterm children and control children differed in their demographic characteristic only with respect to their mean birthweights and gestational ages.

4.3 Intra-examiner reliability

Reliability of the data collection was confirmed by using intra-examiner Kappa statistic. No inter-examiner inconsistency arose as the examination was performed by the one examiner. Based on the intra-examiner Kappa statistic, the observations made on participating subjects were calculated to be substantially reliable. (Overall mean Kappa = 0.716)

Kappa statistics, a reliable means for assessing consistency, had not been previously used in any other studies on enamel defects in the preterm children.

The allocation of teeth in various categories whether it be decayed, filled, hypoplastic and/or showing opacity, were shown to be most

consistent for both the maxillary and mandibular second molars and central incisors. The least consistent judgements were on the maxillary right lateral incisor (Kappa = 0.444) with Kappa value showing it to be moderately reliable and acceptable.

4.4 Prevalence of Enamel Defects

4.4.1 Introduction

Enamel defects were found in 24 of the 25 (96%) of the preterm children at the final examination (Exam III). In contrast, 9 (45%) of the controls were affected (Table 3.5.1 p.52). Therefore, enamel defects affected more preterms than controls and was found to be at a statistically significant level ($P < 0.001$). This was the case also for Exams I and II.

Not only were there relatively fewer control children affected, the number of teeth affected in these control children was even smaller. In Exam III, a total of only 20 (5.0%) of 399 teeth in the control children were affected compared to 191 (65.4%) of 499 teeth affected in the preterm children. Again the pattern was similar for all three examinations with respect to the number of affected teeth. More teeth with enamel defects were found in the preterm children compared to the control children, at statistically significant level ($P < 0.0001$).

Previous studies have reported high prevalences of enamel defects in the VLBW preterm children. Analysis of all available studies with the VLBW preterm children, revealed the percentages of affected children ranged

from 38% for the study by Pimlott *et al* (1985) to 83% as reported by Fearne *et al* (1990) (Chapter 1.2 p.14). Studies by Seow (1986) and Seow *et al* (1987), on earlier batches of preterm derived from the same establishment had a prevalence figure of 79% and 62% respectively. The author found enamel defects prevalences in preterm children of 88%, 94.7% and 96.0% for the three examinations, ie. Exam I, II and III respectively.

These higher prevalences compared to earlier studies are very likely due to the fact that our VLBW children were in the lower birthweight end of the VLBW scale. A total of 18 (69%) out of 26 VLBW children in this present study were below 1,000g. The mean birthweight for the preterm children in this study was 969 ± 243 g, whereas the mean birthweight for preterm children in Seow *et al* (1987) was 1177 ± 193 g. A significant difference of 208g in the mean birthweights, (P-value < 0.0001). This would suggest that on an average the preterms at birth were 21.5% heavier in the Seow *et al* (1987) study compared to the present preterm sample. It has been shown that the prevalence of enamel defects increases with decreasing birthweight (Seow *et al*, 1987). This would thus explain for the higher prevalence in the present sample of very low birthweight preterm.

Interestingly, the study by Fearne *et al* (1990) had a prevalence figure closest to the figures reported in this study. Fearne *et al* (1990) reported a prevalence of 83% in their VLBW group. Unfortunately, the mean birthweight of their VLBW group was unavailable for comparison.

Regarding the prevalence rates of enamel defects in the control children

of this study, again the rates were higher than that reported by Grahnen *et al* (1974); Johnsen *et al* (1984); Seow *et al* (1987). The percentages of affected control children in the present study were 40.4%, 54.5% and 45.0% in Exam I, II and III respectively. Comparing these rates to that reported by Seow *et al* (1987) of only 12%, leaves one pondering as to why the present prevalence rates are much higher. As will be discussed further in later section (Chapter 4.5.2 p.105) the enamel defects observed in the control children were mainly enamel opacities, which can quite easily remain undetected. Here the author had the opportunity to examine the sample three times, and having only a small group of children to monitor, whatever anomalies present were closely scrutinised.

Furthermore, the enamel defects prevalence in control groups of other recent studies were comparable to that of the author. Percentages of affected control children were 37% in the study by Fearne *et al* (1990) and 40% in Mellander *et al* (1982).

Although the percentages reported here for affected control children were 40.4%, 54.5% and 45.0% for Exam I, II and III respectively, the number of teeth affected in the control children was very small. This meant that while many children were affected, the number of affected teeth in each child was small. Only 3.4%, 5.5% and 5.0% of the total teeth in the control children were affected in Exam I, II and III respectively. On the other hand, in the preterm children the number of affected teeth in comparison was very large. In Exam I, II and III the percentages of affected teeth were 17.8%, 23.0% and 38.3% respectively. On average for example, the mean affected teeth for each preterm child was 7.6 teeth/child compared to 1.0 tooth per control, at Exam III.

The percentages of affected teeth in the VLBW preterm and control children have not been previously described in other available studies except for Fearne *et al* (1990) who gave a bar graph representation of the percentages of each type of teeth affected. No actual percentages were quoted in any available studies and thus no comparison could be made. Nevertheless, judging from graphical representation in the Fearne *et al* (1990) study, the trend of more preterm teeth being affected by enamel defect than control teeth was obvious.

4.4.2 Prevalence of the various types of enamel defects.

In the preterm group at Exam I, the percentages of children affected by the various types of enamel defects, i.e. hypoplasia, opacity and hypoplasia with opacity concurrently on the same tooth (HO) were 68%, 48% and 0% respectively. This would mean that hypoplasia was the most prevalent enamel defect seen in Exam I. However, in Exam II and III, the opacity type was more prevalent than the hypoplasia type again with the HO type being least prevalent. Opacities affected 73.7% and 84.0% of the preterm children in Exam II and III respectively whereas hypoplasia affected 52.6% and 64.0% of the children in Exam II and III respectively.

There are several reasons for the increased prevalence of opacities observed at later examinations. The first reason being that a greater percentage of teeth that erupted later in the mouth possessed enamel opacities rather than hypoplasia. Another reason was the finding that teeth not previously observed to be affected with opacities in Exam I were noted to be affected in later examinations. Such changes in enamel defects with time have also been reported by Williams *et al* (1994), based

on photographic evidence in 38 affected teeth of the permanent dentition. These changes are thought to be the result of continuing post-eruptive processes of enamel maturation and interaction with saliva on previously undetected, subsurface enamel defects which may with time become clinically obvious opacities (Seow and Perham, 1990). The third reason was related to the decrease in prevalence of hypoplasia. There were several anterior teeth with enamel hypoplasia located on the incisal edges of these teeth in Exam I. With time these thin incisal edges tended to be worn down thus removing the hypoplastic areas and consequently, not being recorded as teeth affected with enamel hypoplasia in later examinations.

In the affected control children, at all three examinations enamel opacities were most prevalent followed by HO and hypoplasia. Enamel opacities were found in 28.0%, 45.5% and 30.0% of control children in Exam I, II and III respectively. Although a significant portion of controls were affected, the number of teeth affected in these children at each examination was small.

Comparing the various types of enamel defects found in both preterm and control children, enamel hypoplasia affected preterms more than controls in all three examinations at statistically significant levels ($P < 0.001$). Enamel opacities and HO were statistically significant only in Exam III. This would mean that the larger number of preterm children affected with enamel opacities and HO in both Exam I and II were not statistically significant, compared to the controls. This may look deceptive initially giving the impression that preterm and control children are equally affected by opacities and HO at Exam I and II, but

if one were to analyse the data in greater depth by looking at the number of affected teeth in the preterm and control children, the picture will be very different and clearer.

Figs. 3.5.3 and 3.5.4 (p.61) compare the percentages of teeth affected by the various enamel defects. These figures and Table 3.5.3 (p. 63) show clearly that there are significantly more enamel opacities, hypoplasia and HO, on the whole, in the preterm than in the control children ($P < 0.0001$).

In the preterms, prevalence of the various enamel defects according to the number of affected teeth followed the same trend as that of the number of affected children (compare Fig. 3.5.1 p.55 to Fig. 3.5.3 p.61). In Exam I, hypoplasia was slightly more prevalent than opacities, with HO not being observed. In Exam II, opacities were more prevalent, followed by hypoplasia and HO. In Exam III, opacities were most prevalent followed equally by hypoplasia and HO.

Previous literature (Table 1.1 p.15) has already shown that the disturbances in enamel formation as a consequence of prematurity causes the high prevalence of enamel defects seen. Many systemic factors are known to be associated to enamel defects including birth trauma, infections, nutritional disorders, metabolic disorders occurring prenatally, perinatally or postnatally. The pathogenesis however, of enamel defects is still not well understood. Recently, there is evidence to suggest that it may involve the central mechanism of osteopenia (Seow *et al*, 1989), with the possibility of being supervened by direct cellular damage to ameloblasts through infective agents or local trauma.

4.5 Prevalence of dental caries

4.5.1 Introduction

As shown in Table 3.6.1 (p.65) at the commencement of the study, (Exam I) the baseline caries experience was zero for the preterm children (ie. no preterm children were affected) and one control child had caries.

At Exam II, 4 (21%) preterm children had caries experience in contrast to again the one control child as in Exam I with caries experience. Similarly, at Exam III, 7 (28%) preterm children had caries experience compared to 4 (20%) of the control children.

Despite the fact that these differences did not reach statistical difference, there are significant clinical implications. In relation to S.E.S. classes, caries tended to indiscriminately occur in preterm children whereas there was a predominant shift of high prevalence in the low S.E.S. class in the control children. In the dental literature, higher dental caries prevalence is normally associated with lower S.E.S. classes (Disney *et al*, 1992; Bohannen *et al*, 1985; Abernathy *et al*, 1987). Furthermore, it was likely that the control group as a whole may be biased with respect to having more children with caries experience than the normal population. These children may have presented for the study because the parents were aware that dental problems may be present. In short, we may have included more than what should be the likely number of high caries risk children in the control group of that size.

This theory of including greater than the proper proportion high caries risk children in the control group is being supported by the fact that in Exam II, although only one (9%) child in the control group was caries affected, he had 5 carious teeth. This is compared with 4 (21%) preterm children with caries experience in Exam II, with a mean of 2.75 carious teeth per affected preterm child. Again in Exam III, 4 (20%) control caries-affected children had a mean of 7 carious teeth per affected control, in contrast to 7 (28%) caries-affected preterm children with a mean of 2.9 carious teeth per affected preterm child.

Furthermore, judging the caries prevalence using the d.m.f.t scores in this present study may not be accurate. The mean d.m.f.t score again gave the impression that caries experience is almost equal in Exam II for both control and preterm groups. Even more erroneous in Exam III, caries experience was higher in the control children at 1.4 ± 3.2 compared to 0.8 ± 1.5 in the preterm children. The mean d.m.f.t. score is not a good caries indicator particularly when there is a large number of carious teeth being experienced by a small number of children; here in this case we had a small number of children (4 in actual fact) with high caries rate in the control group inflating the d.m.f.t. score for the control group.

It may be more pertinent to examine the percentages of caries-free children. There were 100% of caries-free preterm children at Exam I (baseline) reducing to 79.0% at Exam II and 72.0% at Exam III (final exam). In the controls 96% were caries-free at Exam I (baseline) reducing to 91.0% at Exam II and 80.0% at Exam III (final exam). The author found lesser percentages of caries-free preterms than controls.

However, statistical testing did not show any significant differences between the preterms and controls.

The present study would support the findings of Grahnen and Larsson (1958) only as far as the mean d.m.f. score is concerned. Grahnen and Larsson (1958) found no statistical significant difference in the caries score between their thirty-five paired preterm and matched control groups. However, to simply conclude that caries experience is no different in our present investigation based on the d.m.f.t score alone is premature for the reasons discussed earlier. Rather, the author tends to agree with the view held by Rosenzweig and Sahar (1962), that preterm children with enamel defects were more susceptible to caries.

4.5.2 Decayed (d) and/or filled (f) components of the d.m.f.t. score

The analysis of the decayed (d) and/or filled (f) components may provide an indicator of treatment needs of the children.

The decayed (d) and/or filled (f) components (Table 3.6.2 p.70) showed that at Exam I and II only children with the decayed component were present. In Exam I, (at baseline) one control child had the decayed component. In Exam II, 4 (21.0%) preterm children compared to one (9.1%) control child had the decayed component. The differences observed were statistically not significant. By Exam III, treatment was already rendered to affected children in Exam I and II to result in the filled components. At Exam III more children with active lesions were seen in both groups, 4 (16.0%) preterms and 4 (20.0%) controls.

The number of teeth affected by the decayed component (Table 3.6.3 p.74) at Exam I (baseline) was zero for the preterm and one for the control children. In Exam II there were 11 (2.8%) and 5 (2.3%) teeth with decayed component in the preterms and controls, respectively. By Exam III 10 (2%) and 5 (1.3%) decayed teeth in the preterms and controls respectively, were filled. Here, in Exam III a further 10 (2.0%) and 23 (5.8%) teeth in the preterms and controls respectively, were detected to be decayed. There were statistically more decayed components in the controls than the preterms (P = 0.005) due to the high number of decayed teeth found in several (4 children in actual fact) high caries controls.

These results indicated that children in need of treatment, based on the (d) and (f)-components fell basically into two groups. The first group was comprised of preterm children affected by enamel defects. The second group being comprised of high caries risk control children. These two groups of children at each examination had unmet treatment needs. Parents were informed at each visit of the dental findings. Also, these children were either treated by the author or their private dentist following each examination.

4.6 Distribution of teeth affected by enamel defects and dental caries according to tooth type

Dental caries affected primary second molars most (Table 3.7.1 p.76) with the primary mandibular canines and incisors being spared. The pattern is dissimilar to that of bottle caries in that the maxillary incisors and canines are less frequently affected. In this study only 2% of the

maxillary incisors and canines were involved compared to 18% of the maxillary second molars and 10% of the mandibular second molars. In other words the maxillary incisors and canines were 9 fold less likely to be affected by caries compared to the maxillary second molars and 5 fold less likely compared to the mandibular second molars. Furthermore, the carious surfaces involved usually favour the occlusal third and are closely associated to enamel defects (Chapter 4.8 p.110), with cuspal involvement rather than at pits and fissures, contact points or at gingival third of crowns.

Caries pattern in the preterm had not been investigated prior to this study in the white population.

The author found the pattern of dental caries to be almost similar to that of the distribution of teeth affected by enamel defects except that there were more posterior than incisor teeth affected by caries. Dental caries was mostly found on the second molars, followed by the maxillary first molars. At equal third places, were the mandibular first molars, maxillary incisors and canines. It would seem even though enamel defects are strongly associated to caries susceptibility (Chapter 4.8 p.110)there are also other factors acting upon the posterior teeth to make them more susceptible. These factors may be the occlusal forces and the extent of the enamel opacities on the involved teeth. Enamel opacities found in posterior teeth usually involved at least two-thirds of the occlusal surfaces and these opacities are constantly under direct heavy occlusal stresses in contrast to enamel opacities on maxillary incisors located on the buccal surfaces sheltered from occlusal stresses. Therefore, opacities on the occlusal surfaces of the posterior teeth are more easily

abraded by occlusal stresses than fully mineralised enamel (Suga, 1992) and is thus the most likely reason for the higher number of carious lesions observed on posterior teeth with enamel opacities.

For the control children, teeth most affected by enamel defects were on the canines followed by the maxillary incisors and second molars, which is a reverse pattern of distribution compared to that of the preterm children. Not unexpected was the distribution of dental caries. Dental caries affected first molars the most, followed by the maxillary incisors (- a pattern of caries distribution favouring maxillary teeth that have been exposed to the deleterious effects of the oral cavity for longer).

Other investigators looking at enamel defects and caries in the preterms, have <u>not</u> reported on the distribution of enamel defects and caries. Fearne *et al* (1990), although not providing any data on caries, did report in a graphical form the distribution of teeth affected by enamel defects, according to tooth-type. They found enamel defects most frequently on incisors followed by the molars and canines. Major differences noted were in the prevalences of enamel defects reported for the molars and canines. Fearne *et al* (1990) reported percentages of about 10% for molars and less than 10% for canines (compare these figures to that of 42%-52% for the various types of molars and 30%-32% for the canines in the present investigation). Such direct comparison may not be valid for the reason that the preterm children in the study by Fearne *et al*, (1990) had a mean birthweight of 1400g with 50(45%) children weighing between 1501-2000g. Preterm children in this present study had a mean birthweight of 969g. With the increasing prevalence of teeth affected by enamel defects observed in decreasing birthweight preterm, it is to be

expected that the higher prevalences can only come about by enamel defects affecting other teeth (that is the canines and molars) besides the incisors.

Dental caries in the control children was most frequent in the primary first molars (15%) in both arches, followed by the maxillary second molars (10%) and closely tailed by maxillary incisors (7.6%). This caries pattern is not too dissimilar to that seen in the general population, where teeth being present for the longest in the mouth are more likely to be affected except for the lower anteriors protected by the caries inhibitory properties of saliva.

4.7 Association of enamel defects and dental caries

In the preterm children, the relationship between enamel defects and caries was statistically extremely significant ($P < 0.0001$) with the appearance of caries in Exam II and III (Table 3.8.1 p.79). Caries tended to favour teeth with enamel defects more than teeth without enamel defects.

In the controls, the association of enamel defects and caries is shown not to be statistically significant. This is not surprising as the number of teeth affected with enamel defects was relatively small. This was why the study had specifically chosen VLBW preterm children for the reason that these children tended to show much higher numbers of teeth with enamel defects. For example, 37.7% of preterm teeth affected with enamel defects compared to 5.1% of control teeth in Exam III.

The association between enamel defects and caries had also been attempted by earlier authors. Rosenzweig and Sahar (1962) observed in a cross-sectional study that the preterm children with enamel defects had significantly higher caries prevalence than both control children and preterm children without enamel defects. Unfortunately, no direct correlation was made between the distribution of enamel defects and teeth. It may well be that teeth affected with enamel defects were not the teeth exhibiting caries. Furthermore, no distinction was made between opacity and hypoplasia and in fact hypoplasia as a term was used (instead of enamel defects) to describe all types of affected teeth.

4.8 Dental caries and different types of enamel defects

In the preterm children enamel opacities were the only type of enamel defects found to be associated with dental caries (Table 3.9.1 p.84) at an extremely significant level ($P < 0.00001$). Other types of enamel defects were not associated with caries at any statistically significant level.

This observation is supported by findings in other populations studied. In particular, the study by Duray (1992) on both deciduous and permanent teeth of 206 prehistoric Native Americans on the Libben cemetery site. He classified the enamel defects found, based on the D.D.E. index. A strong positive relationship between demarcated opacities and caries susceptibility was reported by him. This relationship was even stronger in deciduous teeth (Duray, 1990). Even as far back as 1947, Stein reported `chalky teeth' (otherwise known as opacities) to be more

susceptible to caries. This he noted in five mature birth children who had suffered severe illness in the neonatal periods.

The findings by Duray (1992) and Stein (1947) are in contrast to Pascoe & Seow (1994), who noted the HO group as being more closely associated with caries than the opacities alone group, in the Tiwi Aboriginal children studied.

The reason for this, is likely due to the fact that teeth affected with opacities and caries are difficult to be differentiated from teeth affected with opacities, hypoplasia and caries due to the large extent of tooth structure destruction in areas of enamel defects. The author has erred in describing teeth in this situation to be that of opacities and caries whereas in the study of Seow *et al* (1994), the investigators had opted for the latter, diagnosing such teeth as being affected by opacities, hypoplasia and caries.

In the control, no association could be found between the different types of enamel defects and caries for the reason that insufficient numbers of the various enamel defects were present.

4.9 Other factors influencing dental caries

An assessment of the various factors influencing dental caries showed that there was little association between the factors listed in Table 3.10.1 (p.87) and caries experience, with the exception of *Strep. mutans* score and this was only applicable to the control group. The lack of

association between various influencing factors and caries had also been encountered in several other large scale studies done on assessing the individual caries risk factors (Leverett *et al*, 1993a; Leverett *et al*, 1993b; Beck *et al*, 1992; Graves *et al*, 1992; Disney *et al*, 1992; Graves *et al*, 1991).

4.9.1 *Strep. mutans* score

Judging from the distribution of *Strep. mutans* scores obtained for the preterm children (both caries-present and caries-free groups), one could deduce that the score bears no relationship to caries experience.

Having said that, interestingly, in the control groups, a trend was observed between the *Strep. mutans* score and caries experience. Caries-free controls tended to have a low score and caries-present controls having higher scores. In this study 10 out of 11 caries-free controls had a score of 0 whilst all caries-present controls had a score of 2 or 3. A polarisation of scores could be seen. Caries prevalence and salivary *Strep. mutans* had also been demonstrated in young children in studies of Crall *et al* (1990) and Fujiwara *et al* (1991).

4.9.2 Plaque score

Examining the plaque scores reveal that the scores were not related to caries and experience. The majority of caries-present and caries-free preterm had lower plaque scores. In the controls, little could be made out of the distribution of the plaque score, although the majority of caries-free controls had a score of 1.

4.9.3 Toothbrushing history

The mean brushing frequencies in both the preterm and controls groups were not related to caries experience. No pattern in either groups could be seen. This is not surprising especially when the data collected was based on parents account of the frequency of brushing which need not necessarily be accurate.

4.9.4 Fluoride exposure

Fluoride exposure and caries and caries experience were not statistically associated in either the control or preterm children. However, it could be seen that only one out of 7 (ratio is 1:7) control children with previous history of fluoride exposure had caries compared to 4 of the 16 (ratio is 1:4) controls without fluoride exposure experiencing caries. This may mean that control children with a history of fluoride exposure were less caries prone.

This conventional thinking does not apply to the preterm children investigated. 5 of the 15 (ratio of 1:3) preterm children with fluoride exposure experienced caries whereas 2 of the 11 (ratio of 1:5.5) preterm children without fluoride exposure experienced caries. It would seem that fluoride exposure did not reduce the caries risk in the preterm children. In fact, the non-fluoride exposed preterm group had less caries experience. This can be explained based on the argument that exposure to fluoride may be difficult to assess. Accurate allocation of children into fluoride exposed and non-fluoride exposed groups would be close to being impossible. Fluoride can be derived from many sources.

Besides prescribed sources in the form of fluoride drops, tablets, gels and mouth washes, other sources include the inadvertent ingestion of toothpaste and manufactured foods containing fluoride. Australian made infant formulas and canned baby foods may vary in fluoride concentration (Messer, 1994), as these fluoride levels are not regulated. These hidden fluorides in various foods consumed are very bioavailable, thus making the classification of children into fluoride exposed and non-exposed groups arbitrary and very challenging. It was calculated that in Australia, a 2-year-old in a fluoridated area would consume about 1.10-1.20 mg fluoride per day from all sources and in a non-fluoridated area, this figure would reduce by only 0.2 mg per day (Riordan, 1994).

4.9.5 Sugar intake

The mean daily frequencies in both preterm and control children were fairly well distributed over the 4 classes of mean frequency. No relationship to caries experience could be found in the children studied.

4.9.6 Socioeconomic status (S.E.S.)

Our results tend to indicate that caries-present preterm children are susceptible to caries regardless of social class. 4 caries-present preterms belong to the S.E.S. I group, 1 caries-present preterm in the S.E.S. III and 2 caries-present preterms in S.E.S. IV (Table 3.10.2 p.90). In the control children, the trend is very different to that of the preterms but similar to that expected of the general population. Caries tend to favour children of the lower S.E.S. 4 out of the 5 caries-present controls were from S.E.S. III and IV (Table 3.10.2 p.90).

Similar results as for the control children had recently been reported by Disney *et al* (1992), Bohannen *et al* (1985) and Abernathy *et al* (1987). In these studies the education level of the head of the household (H.H.D.) was used to establish S.E.S. for the reason that they found an even stronger association between caries and H.H.D. The author felt that questions relating to H.H.D. may be difficult to elicit and therefore type of occupation of parents was obtained instead.

Thus in general, we observed little association between caries and the various influencing risk factors. One would very easily put this down to the small number of participants in this study, but several large scale studies have concluded similarly (Disney *et al*, 1992, Bohannen *et al*, 1985 and Abernathy *et al*, 1987).

4.10 Type of Occlusion

Occlusal variables investigated showed little statistical differences between the preterm and control children. Using the canine relationship as a descriptor of dental antero-posterior relationship, in the preterm group the majority of children, 21 (80.8%) were Cl I. In the control sample, again the majority of children 15 (55.6%) being Cl I.

Unilateral lingual cross-bites were observed in near equal frequency in the preterm and control children, 15.4% and 14.8% respectively. No buccal cross-bites were observed. Of interest were the findings of lower incisor retroclination in 2 (7.7%) preterm children and crowding of the lower incisors in 3 (11.5%) preterm children, all with dental Class I

molar and canine relationship. It would seem that prematurity per se did not alter the prevalence of dental malocclusion in these children. Previous studies on preterm children had little information on the occlusal variables of the preterm children.

Chapter 5 Conclusions and Recommendations for future studies

This controlled investigation is the first longitudinal study available on the developmental enamel defects and caries in the preterm children. The results demonstrated higher prevalence of developmental enamel defects in the very low birthweight (VLBW) preterm children compared to full-term controls. The author found prevalence rates at Exam I, II and III of 88.0%, 94.7% and 96.0% in the preterms in contrast to 40.0%, 54.5% and 45.0% in the control children respectively (P-values < 0.05).

Not only were there more preterm children affected, the number of teeth affected were markedly increased. Percentages of teeth affected at Exam I, II and III were 17.8%, 23.0% and 38.3% in the preterm children in comparison to 3.4%, 5.5% and 5.0% in the control children (P-values < 0.00001)

The mean d.m.f.t. scores at Exam I, II and III were 0, 0.6 ± 1.4 and 0.8 ± 1.5 for the preterm children, in comparison to 0, 0.5 ± 1.5 and 1.4 ± 3.2 in the control children respectively (P-values > 0.05). Judging caries prevalence using the d.m.f.t. scores may not be accurate because of the inflated d.m.f.t. scores for the control group as a consequence of 4 control children with high caries rate. Caries in the control group were found only in these 4 high caries rates children with the rest of the control group not affected by caries. It may be more pertinent to consider caries prevalence based on either the percentages of children with caries experience or the percentages of caries-free children. At Exam I, II and III the percentages of children with caries experience were 0%, 21% and 28% in the preterms in comparison to 4%, 9% and 20% in the control children respectively ($P>0.05$). However, the differences between the numbers of preterm and control children experiencing caries

did not reach statistical significance, as a result of the small numbers of children involved.

Analyses of caries risk factors investigated in this study found enamel defects to be the only underlying predisposing caries risk factor significantly associated with caries (P-value < 0.00001). *Strep. mutans* score did suggest a possible association in the control children. Other caries risk factor considered in this study did not reveal any association (P-values > 0.05). These included brushing frequency, fluoride exposure, daily frequency of sugar intake and plaque score.

Of interest also was the fact that caries affected the preterm children of all social classes indiscriminately, rather than favouring lower social classes as in the full-term controls. This finding together with the highly significant (P-value < 0.00001) association of enamel defects and caries in the preterm have important clinical implications. It would suggest that the underlying enamel defects present in the preterm children obviate the effects of routine caries preventive measures including dietary and oral hygiene habits usually practised by the higher social classes. These measures would have been effective otherwise, in the normal situation where the children were unaffected by enamel defects. Preterm children with a high prevalence of enamel defects (resulting in an increased caries risk) would therefore require more comprehensive professional dental services.

Furthermore, this study noted a predilection for caries to occur in the posterior teeth of the preterm children. Dental caries was most frequently found on primary second molars (18% of maxillary and 10% of

mandibular), followed by the first maxillary molars (4%) and with caries on the maxillary primary first molars, maxillary incisors and maxillary canines at the same frequency of 2%. The primary mandibular canines and incisors were not affected by caries.

High percentages of maxillary and mandibular second molars were affected with enamel defects as were the maxillary incisors. It would seem even though enamel defects are strongly associated to caries susceptibility there are also other factors acting upon the posterior teeth for caries to favour these teeth more. These may be the occlusal forces and the extent of the enamel opacities on the involved teeth. Enamel defects found in posterior teeth usually involved at least two-thirds of the occlusal surfaces and these defects are constantly under direct heavy occlusal stresses in contrast to enamel defects on maxillary incisors located on the buccal surfaces sheltered from occlusal stresses. Therefore, defects on the occlusal surfaces of the posterior teeth are more easily abraded by occlusal stresses than fully mineralised enamel (Suga, 1992) and is the most likely reason for the higher number of caries on posterior teeth affected with enamel defects.

The findings described in this thesis, need to be interpreted with caution due to the small number of subjects available. These findings await large scale studies for confirmation.

Recommendations for future longitudinal studies are:

1. Recruitment of a greater number of preterm and control children

2. Inclusion of full mouth photographic records and impressions. This

may prove difficult especially in the light of the age of these participants (about 2 years old) at the commencement of the study.

3. Closer intervals of monitoring perhaps three monthly or requesting parents to take their child in for inspection upon eruption of new posterior teeth prior to the rapid onset of caries, should caries occur.

4. A longer period of follow-up.

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APPENDIX I

Letter to low birth weight subjects

DEPARTMENT OF DENTISTRY

THE UNIVERSITY OF QUEENSLAND
Dental School
Turbot Street
Brisbane Qld 4000 Australia
Telephone (07) 365 8111
International +61 7 365 8111
Facsimile (07) 365 8199

Dear

We invite you to participate in a dental examination, now being offered to children of the Growth and Development Clinic at the Mater Hospital. The examination is part of a research study conducted by the division of Children's Dentistry, Dental School, University of Queensland. The aim of the study is to detect abnormalities of the teeth associated with low birth weights in children. Early diagnosis of these abnormalities may lead to the prevention of further problems. Participation will require dental check-ups at approximately 2, 3 and 4 years old.

Participation in the study involves only a routine dental clinical examination of your child once a year at the University Dental School for a period of approximately 2 years. These examinations which are provided free of charge, will be carried out by Associate Professor W K Seow, Dr P Y Lai and Dr L McAllan, University dentists who specialise in dentistry for children. No x-rays will be taken unless a particular need is evident. You will be advised of any treatment required.

A consent form is enclosed. If you are willing to participate in the study, please keep the appointment made below for the dental examination of your child. Would you please phone us to confirm the appointment or if you are unable to attend, or if you have any queries regarding this study. (Please ask for Miss Barbara Taylor, Tel. No.: 365 8059)

Thank you for your cooperation.

Yours sincerely

W Kim Seow
Associate Professor in Dentistry for Children
University of Queensland

An appointment has been made for: _____

Date: _____

Time: _____

Place: Clinic 6, Dental School, University of Queensland, Turbot Street, Brisbane. (Please use entrance opposite SUNCORP Theatre, Take lift to second floor).

APPENDIX II

Letter to control subjects

DEPARTMENT OF DENTISTRY

THE UNIVERSITY OF QUEENSLAND
Dental School
Turbot Street
Brisbane Qld 4000 Australia
Telephone (07) 365 8111
International +61 7 365 8111
Facsimile (07) 365 8199

Dear

We invite you to participate in a dental examination, now being offered to children born at the Mater Hospital between 1989–1992. The examination is part of a research study on the dental health of normal children conducted by the division of Children's Dentistry, Dental School, University of Queensland.

Your child has been selected for this study, and we hope you will consent to this study.

Participation in the study involves only a routine dental clinical examination of your child once a year at the University Dental School for a period of approximately two years. These examinations which are provided free of charge, will be carried out by Associate Professor W K Seow, Dr P Y Lai and Dr L McAllan, University dentists who specialise in dentistry for children No X-rays will be taken unless a particular need is evident You will be advised of any treatment required.

A consent form is enclosed. If you are willing to participate in the study, please keep the appointment made below for the dental examination of your child. Would you please phone us to confirm the appointment or if you are unable to attend, or if you have any queries regarding this study. (Tel: 365 8059)

Thank you for your cooperation.

Yours sincerely,

W K Seow
Associate Professor in Dentistry for Children
University of Queensland

An appointment has been made for _____

Date: _____

Time: _____

Place: Clinic 6, Dental School, University of Queensland, Turbot Street, Brisbane (Please use entrance opposite SUNCORP Theatre Take lift to second floor)

APPENDIX III

Letter for follow-up examination

DEPARTMENT OF DENTISTRY

THE UNIVERSITY OF QUEENSLAND
Dental School
Turbot Street
Brisbane Qld 4000 Australia
Telephone (07) 365 8111
International +61 7 365 8111
Facsimile (07) 365 8199

Dear

We invite you to participate in the follow-up dental examination on your child. The final dental examination will be performed in 1994. These examinations form part of a study on the dental health of children conducted by the Division of Children's Dentistry, Dental School, University of Queensland.

Participation in the study involves only a routine dental clinical examination of your child once a year at the University Dental School for a period of approximately two years. These examinations which are provided free of charge, will be carried out by Associate Professor W K Seow, Dr P Y Lai, Dr L McAllan, University dentists who specialise in dentistry for children. No X-rays will be taken unless a particular need is evident. You will be advised of any treatment required.

We look forward to seeing your child once again at the appointed time below. Would you please phone us to confirm your appointment, or if you have any queries. (Tel: 3658059)

Thank you for your cooperation.

W K Seow

Associate Professor in Dentistry for Children
University of Queensland

An appointment has been made for: _____

Date: _____

Time: _____

Place: Clinic 6, Dental School, University of Queensland, Turbot Street, Brisbane. (Please use entrance opposite SUNCORP Theatre. Take lift to second floor).

APPENDIX IV

Letter for final follow-up examination

DEPARTMENT OF DENTISTRY

THE UNIVERSITY OF QUEENSLAND
Dental School
Turbot Street
Brisbane Qld 4000 Australia
Telephone (07) 365 8111
International +61 7 365 8111
Facsimile (07) 365 8199

Dear

We invite you to participate in the final follow-up dental examination on your child. The examination is part of a study on the dental health of children conducted by the division of Children's Dentistry, Dental School, University of Queensland.

Participation in the study involves a simple inspection of the teeth of your child at the University Dental School. The examination which is provided free of charge, will be carried out by Associate Professor W K Seow, Dr L McAllan or Dr P Y Lai, University dentists who specialise in paediatric dentistry. No X-rays will be taken unless a particular need is evident. You will be advised of the dental findings and will have the opportunity to discuss any aspect of oral health with the examining dentist. Routine dental treatment for your child may be offered with no fees, at our University Dental Clinic, if you so desire.

We look forward to seeing your child once again at the appointed time below. Would you please phone us to confirm the appointment or if you are unable to attend, or if you have any queries regarding this study. (Tel: 365 8059)

Thank you for your cooperation.

Yours sincerely,

W Kim Seow, DDSc, PhD, FRACDS
Associate Professor in Dentistry for Children

An appointment has been made for _____

Date _____

Time _____

Place: Clinic 6, Dental School, University of Queensland, Turbot Street, Brisbane (Please us entrance opposite SUNCORP Theatre, Take lift to second floor)

APPENDIX V

Consent form

FACULTY OF DENTISTRY

THE UNIVERSITY OF QUEENSLAND
Dental School
Turbot Street
Brisbane Qld 4000 Australia
Telephone (07) 365 8111
International +61 7 365 8111
Facsimile (07) 365 8199

CONSENT FORM

Title: Dental decay and enamel defects study

Investigators: W.K. Seow, P.Y. Lai

Study Description:

The study will investigate the prevalence of dental decay and abnormalities of teeth in young children. The results of the study will improve the understanding of these conditions and help in the implementation of effective preventive dental health programmes.

Participation in the study involves the completion of a simple questionnaire by you, and your consent for your child to a simple, routine dental examination at the University Dental School. No dental X-rays will be taken as part of the study. The results of the dental examination will be explained to you.

Child's Name (Block Letters): _____

I consent to my child for routine dental examination at yearly intervals for a period of 2-3 years by a dentist at the University of Queensland Dental School.

I am aware that I am free to withdraw my child from the study at any time. I understand that all information collected will be safeguarded in a confidential manner.

I, _____ (parent/guardian) have read the description and understand the nature of the study

Signed: _____

Date: _____

APPENDIX VI

Post natal Medical History form

POST NATAL HISTORY

I. Medical

1. Current doctor/pediatrician:

2. Current medication:

3. Is there any reason for child to be suspected of being at risk to having AIDS or any disease related to AIDS?

4. Has your child ever had – (Please tick Yes or No)

	Yes	No
(a) Heart disorder	☐	☐
(b) Rheumatic fever	☐	☐
(c) Bleeding problems	☐	☐
(d) Blood transfusion	☐	☐
(e) Anaemia	☐	☐
(f) Bronchitis	☐	☐
(g) Asthma	☐	☐
(h) Jaundice	☐	☐
(i) Hepatitis	☐	☐
(j) Diabetes	☐	☐
(k) Epilepsy	☐	☐
(l) Allergy to any medication	☐	☐
(m) Chicken Pox	☐	☐
(n) Other developmental problems, medical syndromes or major illnesses If yes Please specify	☐	☐

5. Immunizations

	Yes	No	?
Triple Antigen	☐	☐	☐
Tetanus Booster	☐	☐	☐
Measles	☐	☐	☐

APPENDIX VI (Cont'd)

Dental History form

II DENTAL HISTORY

1. Any previous visit to Dentist? No ☐ Yes ☐

If yes: a) Type of treatment: Check up ☐ Extractions ☐
Topical Fluoride ☐ Others ☐
Fillings ☐ Specify _____
Cleaning ☐ _____

b) During treatment was any of the following used:
Local anesthetics ☐
general anesthetics ☐
relative anesthetics ☐

c) Any difficulty and/or complications with the above treatment: Yes ☐ No ☐

2. Fluoride History

a) Fluoride Supplements Yes ☐ No ☐

If yes, When started: _____

Form, Dosage and Frequency _____

Lived in Brisbane since born Yes ☐ No ☐

If not, in fluoridated area _____

3. Oral Hygiene Habits

a) When Started: _____

b) Brushing: Toothbrush ☐ Frequency/Day ☐ Parents help ☐
Cotton buds ☐ Frequency ☐ Parents help ☐
With toothpaste ☐ Amount used: 1cm, 2cm or 3cm
Is toothpaste swallowed Yes ☐ No ☐

c) Fluoridated water supply Yes ☐ No ☐

4. **Dental Trauma** Yes ☐ No ☐

If Yes, please provide details _____

APPENDIX VI (Cont'd)

Maternal History form

MATERNAL HISTORY

Maternal age at birth: _____ Yrs

Fluoride in pregnancy: ☐ Fluoridated Water Supply

☐ Supplements

Time started: (circle)

1st 2nd 3rd

Duration: _____ yrs _____ mths

Others: _____

Complications during pregnancy (e.g. trauma, infections):

Medications during pregnancy (type and duration):

Smoke during pregnancy Yes ☐ Quantity/day: _____ Stick(s)

No ☐ _____ Packet(s)

Others: _____

Dental check-up during pregnancy Yes ☐ Frequency

No ☐

Emergency only ☐

Birth complications:

APPENDIX VII

Personal Detail form

PERSONAL DETAILS

Patient's Surname _____ First Name and Initial(s) _____

Address: _____

_____ Postcode: _____

Telephone: _____

Changed Address: _____

_____ Postcode: _____

Sex: Male ☐ or Female ☐ Date of Birth: _____

Dates of dental examination Age at Exam: _____ (Year) (Month)

1. _____
2. _____
3. _____
4. _____

Fathers Name: _____

Occupation: _____

Mothers Name: _____

Occupation: _____

Some helpful suggestions in completing this FOOD DIARY:

1. Choose THREE successive days.
2. Make at least ONE of these days a Saturday or a Sunday.
3. Record EVERYTHING that you eat or drink.
4. Provide as much INFORMATION as possible.
5. Describe AMOUNTS in everyday terms such as cup, tablespoon, slice etc.

REMARKS and RECOMMENDATIONS:

FOOD GROUPS		MINIMUM DAILY SERVINGS (MDS)	EVALUATION: SERVINGS PER DAY DAY 1	DAY 2	DAY 3	SUMMARY Average	Average minus MDS
MILK and CHEESE	CHILDREN	3					
	TEENS	4					
	ADULT	2					
MEAT and OTHER BODY BUILDERS	EVERYONE	2					
FRUIT and VEGETABLES	EVERYONE	4					
BREAD and CEREALS.	EVERYONE	4					
FAT							
FLUIDS							

SUGAR FREQUENCY		NUMBER PER DAY DAY 1	DAY 2	DAY 3	SUMMARY TOTAL	Average per Day
IN SOLUTION	During meals					
	End of meals					
	Between meals					
IN RETENTIVE FORM	During meals					
	End of meals					
	Between meals					

FOOD DIARY

and

DIET EVALUATION

PATIENT

DATE

Your Dentist: _____

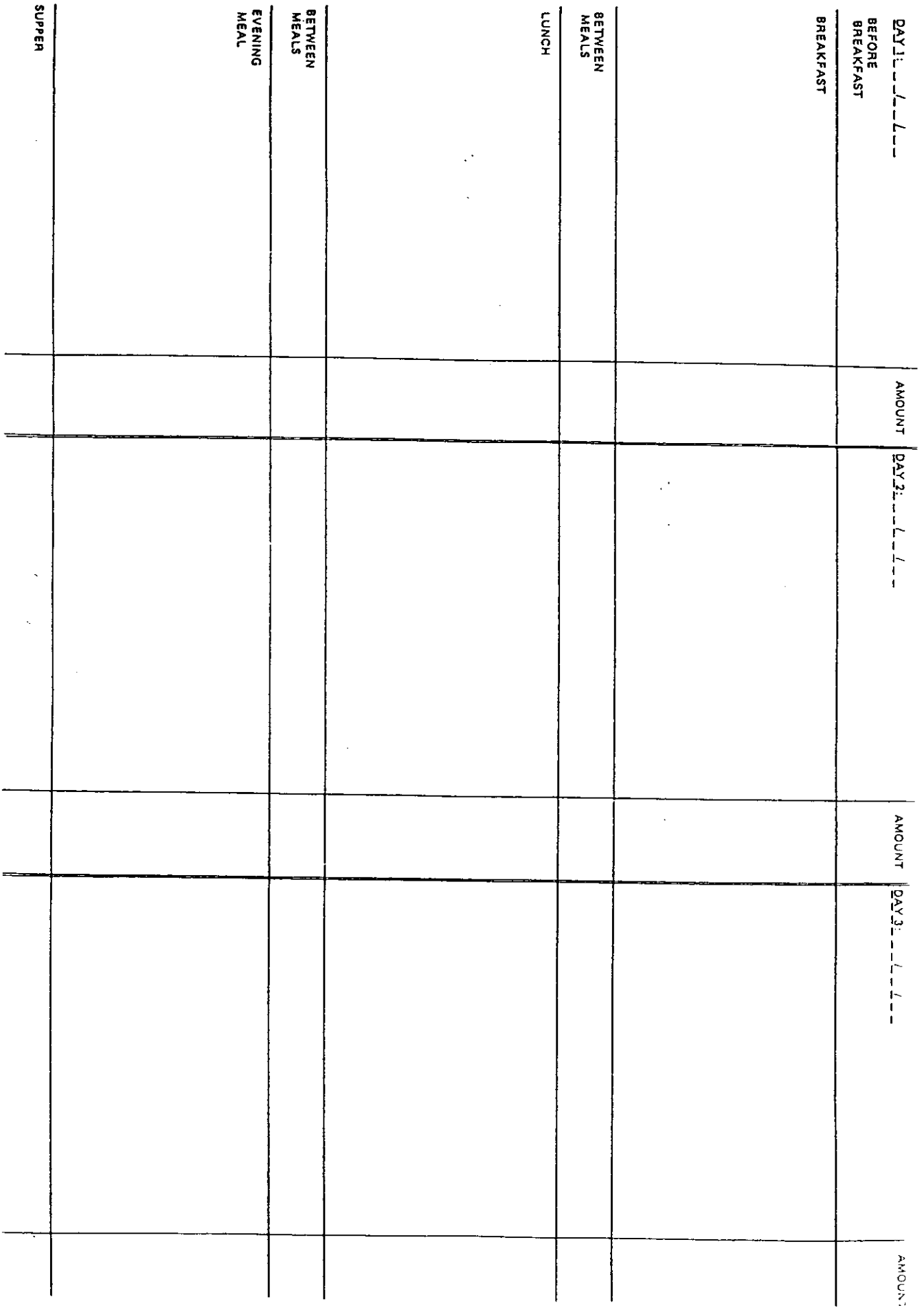
THE UNIVERSITY OF QUEENSLAND

DENTAL SCHOOL
TURBOT ST.
BRISBANE. Q. 4000

APPENDIX VIII

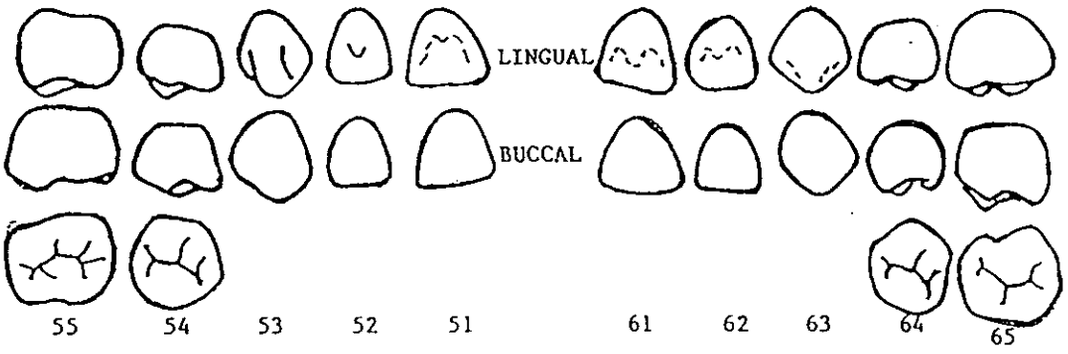
Food Diary and Diet Evaluation form

APPENDIX VIII (Cont'd)

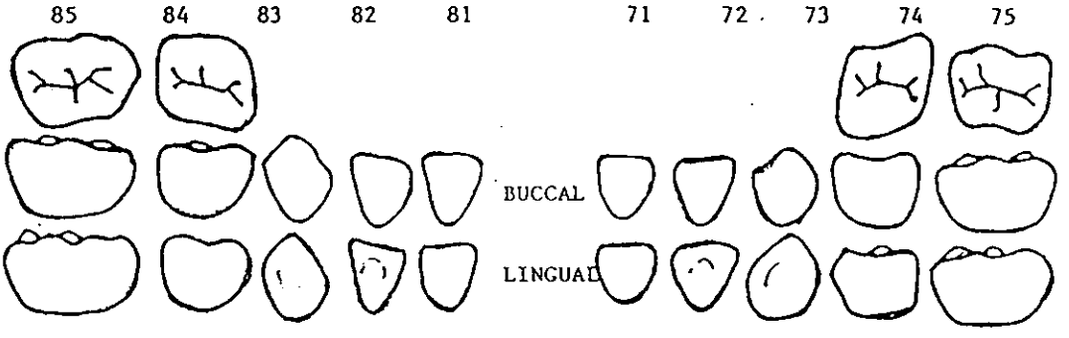


APPENDIX IX

Dental Examination Chart



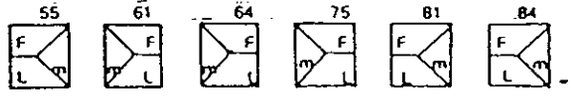
CIRCLE ! TEETH PRESENT



PLAQUE INDEX.

Date / /

Plaque Index



Total scores / Total surfaces = P.I.

APPENDIX X

Occlusion and Digit Habit Chart

Occlusion:	Incisal Relationship	O/J	_____ mm
		O/B	_____ %
	Molar E's	Distal step	_____
		Mesial step	_____
		Flush term plane	_____
Digit Habit:	Night time only		_____
	All day and night		_____